THE
LARYNGOSCOPE.

VOL. XLIX NOVEMBER, 1939. No. 11

# A REVIEW OF THE AVAILABLE LITERATURE ON THE PHARYNX AND PHARYNGEAL SURGERY FOR 1938.

DR. FRANCIS E. LEJEUNE, New Orleans.

## BACTERIOLOGY.

It is interesting to note that in a summary of the observations made by Wirth and Braun[1] on the comparative bacteriology of the epipharynx, the authors state that even during health the epipharynx usually contains numerous micro-organisms common to the oral cavity, particularly the streptococci and Gram negative cocci belonging to the micrococcus catarrhalis group. It is thought the presence of numerous pathological micro-organisms such as hemolytic streptococci, pneumococci and influenza bacilli in the nasopharynx indicates a local infectious process. The exact identification and their classification into pathological and nonpathological forms is considered to be absolutely necessary. The authors have further observed that in the same person, the epipharyngeal flora is not always the same as the nasal or tonsillar flora. In different persons the flora varies greatly. The results of cultures from the epipharynx are generally more reliable than those obtained from the nose and the tonsils. In acute infections the epipharynx as a rule yields a pure culture of the pathogenic organisms, and even after the subsidence of all clinical symptoms of an acute infection, pathogenic organisms, particularly the hemolytic streptococcus, frequently remain on the mucous membranes of the respiratory tract for a considerable period of time. For this reason these patients should be kept away from other patients for a while after the subsidence of fever.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Nov. 23, 1939.

## INFLAMMATORY DISEASE OF THE PHARYNX.

Burghi[2] emphasizes the importance of nasopharyngeal infections and believes their many complications may involve any of the organs. Repeated infections of the nasopharynx will certainly produce a chronic infection and, as Schenck[3] so aptly states, chronic pharyngitis is not always due to constant bathing of inflammatory material from the sinus above or the lung below. He further states that hyperplasia and chronic inflammatory changes in the lymph nodules of the pharyngeal wall are the principal pathological alterations found in chronic pharyngeal infections. Electrocoagulation with administration of iodides is an effective temporary control for the progressive alterations of the lymph tissue in chronic pharyngitis. This not only affords local relief if pharyngeal symptoms are present, but also in a surprisingly large number of cases with such systemic disturbances as has been mentioned, beneficial results were obtained when the removal of other possible foci seemed in vain.

O'Connor[4] studied a series of 59 cases suffering from a low grade fever, all of whom had been subjected to exhaustive examinations, both clinical and laboratory; and yet the cause continued undiscovered. In many of these cases, while the sinuses and oropharynx had been thoroughly investigated, the nasopharynx had escaped attention and frequently enough it offered a solution to the problem. In the cases reported, besides the fever, the most common complaints were fatigue, headache, loss of weight and muscular aches. In all, inflammation of the nasopharynx of varying degrees was found. In many, it was localized to the remains of the pharyngeal tonsil. In others, a diffuse inflammation of the whole nasopharyngeal area was present. A third group showed a localized inflammatory area on one wall or the other. Cultures showed the predominating organisms were streptococcus viridans, pneumococcus type IV, streptococcus hemolyticus and pneumococci type III. In 46 per cent, a pure culture of varying organisms was obtained. Local applications of silver nitrate of varying strength was used, followed by a generous application of 2 per cent aqueous gentian violet or methylene blue; 0.5 per cent colloidal iodine, followed by the dye, was used in those cases which did not respond to the above treatment. If this failed, an autogenous filtrate was made and applied to the area. In

most of the cases, six to 12 treatments were necessary to eradicate the infection. Eradication of the infection was effected in approximately 80 per cent of the cases.

Bajkay[5] states that while adenoid vegetations are found most frequently in children and not uncommonly in young adults, it is, however, unusual to detect them in persons over 30 years of age. Only four or five such cases are reported in the literature, and he adds two more showing hypertrophy of the pharyngeal tonsil; one in a woman, age 51 years, and one in a man, age 33 years. The symptoms complained of were obstruction to nasal breathing, dull voice, impaired hearing, otalgia and nervous disorders.

That exophthalmic goitre has a close relationship to pharyngeal infections is a thought expressed by King.[6] In reviewing a small series of cases he noted certain salient features. All of the cases had attacks of tonsillitis, and bacterial examination of the tonsils showed a great preponderance of hemolytic streptococcus. The incidence of tonsil infections in 200 cases of exophthalmic goitre is considerably higher than in control groups of an equal number of cases with tuberculosis, syphilis and cancer, respectively.

Banyai[7] advocates the use of cod liver oil for the local treatment of tuberculosis of the pharynx. Cod liver oil has a healing effect when applied externally to cuts, abrasions and ulcers of the skin. It is believed by some investigators that cod liver oil has some inhibitory effect on the growth of tubercle bacilli as well as on the general bacterial flora of wounds. With these observations in mind, the effect of local applications of cod liver oil to tuberculous ulcers of the pharynx and larynx was studied. The treatment consisted of simple spraying of ordinary cod liver oil on the ulcerated surfaces.

Richards[8] reviewed a series of 162 cases of retropharyngeal abscess observed at the Children's Hospital in Boston during the past 10 years. Except in cases of dire urgency, he strongly advocates a lateral Roentgenogram of the neck for diagnostic purposes. Inspection and palpation are used routinely in the examination of the cases, yet both have failed to establish a positive diagnosis at times. The chief symptoms were dyspnea, dysphagia, swollen neck, sore throat and fever. The treatment is incision and drainage with the patient preferably in the

Rose position. In 115 cases, the outcome was recorded as uneventful. The mortality was 7.4 per cent. Richards warns that sudden severe hemorrhage, often recurrent, has been one of the most dreaded complications of retropharyngeal abscess. In three such cases it proved fatal. Ligation of the carotid is the only method of successfully controlling these severe hemorrhages.

A spontaneous hemorrhage from a pharyngeal abscess in a child with a tendency to hemophilia resulted in death. Laurent[9] reports such a case and describes a large abscess on the posterior pharyngeal wall, having its centre at about the level of the middle of the epiglottis. The original diagnosis was nasal diphtheria. A soft swelling was noted on the right side of the neck between the angle of the jaw and the sternomastoid muscle. The overlying skin was movable and not reddened. No membranous exudate was noted in the throat but streaks of blood could be seen on the posterior pharyngeal wall. The immediate cause of death was asphyxia from inhalation of blood. In discussing this case, Laurent points out that the abscess may be peritonsillar, retropharyngeal or parapharyngeal. This last type is the one most commonly associated with hemorrhage. When the source of the blood is found it is usually due to an erosion of one of the large vessels of the neck, the internal carotid most frequently, the external carotid and the internal jugular vein less commonly.

Retropharyngeal abscess is a rare condition in adults. Gerocarni[10] believes it is most often due to tuberculosis of the cervical vertebrae, and rarely it may occur as the result of a tuberculous lymphadenitis. All of these types of cases have a chronic course, while the acute cases are caused by peritonsillar abscess, mastoiditis or foreign bodies. Liebermeister[11] claims that the presence of retropharyngeal abscess cannot always be recognized by inspection, especially if all the throat organs are red and swollen. In order to make an accurate diagnosis, palpation of the pharyngeal wall is essential. Merklin[12] discusses retropharyngeal abscess with fistulization into the external auditory canal. Kecskes[13] discusses the pathology and therapy of pre-epiglottic phlegmon. This condition is the same as peritonsillitis lingualis phelgmonosa. French authors speak also of phelgmon of the thyreoglossoepiglottic space. The symptoms are described accurately and the diagnosis is

confirmed by the laryngeal mirror. The abscess is usually superficial but sometimes if it is deep it may spread to the genioglossi hyoglossus and mylohyoideus; and further extension might cause a phlegmonous glossitis. On account of the edema, patients should be hospitalized and the abscess incised intrapharyngeally as soon as possible.

### METHODS OF EXAMINATION.

In discussing the methods for the examination of the nasopharynx, Ciurlo[14] comments on the salpingoscope introduced by Valentine, and on direct pharyngoscopy and the use of the pharyngoscope as compared to the new method which he presents. It consists of a speculum-like instrument which he calls the tube-spatula. It is more efficaciously used with the soft palate and the pharynx anesthetized. Hauser and Brownell[15] believe that any person, regardless of age, with a persistent cervical swelling should have a careful examination of the nasopharynx, and biopsy of any suspicious tissue should be performed. There may be early erosion of the base of the skull or invasion of the sphenoid sinus with resultant headache. Consequently, it becomes a highly desirable practice to examine in a routine manner the nasopharynx of every patient who suffers from unexplained headaches.

### DYSPNEA OF PHARYNGEAL ORIGIN.

An unusual case of dysphagia is reported by Abramowicz.[16] A man, age 48 years, complained of slight difficulty in swallowing for two years. He had a sensation of a foreign body at the back of the throat on the left side. There was no acute pain but occasionally a twinge of shooting into the left ear. At one time the case was regarded as one of cancer; at another, a neurosis. There was no physical sign to indicate any oral pathology beyond a slight asymmetry of the soft palate. Digital examination revealed a bony projection which ended at the lower pole of the tonsil. This he diagnosed as an elongated styloid process, which was confirmed by radiological examination. The patient admitted that pressure over the bony proliferation corresponded to the area where he had felt a foreign body. The author discusses the various views which have been advanced to explain the genesis of this anomaly. Although the generally accepted treatment of this condition

is referred to, there is no mention of the treatment which was carried out in this particular case.

Johnson[17] considers certain aspects of dysphagia associated with anemia and says that the use of tobacco and alcohol are the most common sources of irritation to the oral and pharyngeal mucosa, although dental caries is always a considerable factor. Because the male particularly has indulged excessively in the use of alcohol and tobacco, the incidence of malignancy is so much greater in this sex than in the female. In fact, the presence of carcinoma in the female has often been used as an argument against the validity of Virchow's theory of irritation. While this theory holds for the male, carcinoma of the mouth, pharynx and esophagus in women has recently been observed to be etiologically dependent not upon irritation but rather upon an achlorhydria or hypochromic anemia with the resultant changes in the mucosa of the pharynx, mouth and esophagus.

Anemia in women associated with dysphagia was observed by Plummer in 1914. Vinson, in 1922, augmented Plummer's findings and since then the condition has been called the Plummer-Vinson syndrome. The symptoms are: *1.* a hypochromic anemia; *2.* dysphagia; and *3.* atrophic changes in the lips and oral mucosa and a smooth tongue. Cracking or fissures of the corners of the mouth, early loss of the teeth, changes in the nails and splenomegaly are often observed. The syndrome usually occurs between 15 and 20 years of age, sometimes later, but never after 50 years. The dysphagia is probably due to atrophy of the mucosa in the hypopharynx and atony of the muscles of deglutition. Most frequently it can be discovered that the diet is low in iron.

### CONGENITAL STENOSIS.

Childrey[18] reminds us that few cases of congenital stenosis have been reported. Most of the lesions are acquired. Borghesan,[19] however, reports a congenital stenosis seen in a newborn infant and described by him as a total bilateral choanal osseous diaphragm. Operation was performed a few hours after birth. A triangular opening was made on each side into the diaphragm. Four months later, the opening persisted but it was thought that surgical intervention would be again necessary in years to come.

Childrey believes that stenosis of the pharynx is produced mostly as a result of infectious diseases and that syphilis is the greatest offender. The other common cause is trauma. Two cases of cicatricial stenosis following tonsillectomy and adenoidectomy are reported. Possibly the reason for the rarity of this condition is explained by the mobility of the palate and the faucial pillars. In order to cause stenosis, the inflammatory process must be severe enough to cause fixation of the soft palate for some time, considerable fibrosis and temporary destruction of the epithelium. The symptoms vary according to the amount and density of the scar tissue and the degree of obstruction. The voice is usually flat and lacking in resonance. If the uvula is missing and a small opening remains in the nasopharynx the voice may not be markedly affected, especially if there remains some mobility of the soft palate. These patients complain of mouth-breathing, dryness of the pharynx, irritation of the larynx and discomfort in swallowing. The symptoms are aggravated by colds. When the nasal secretions accumulate in the nasopharynx, there is impairment of the sense of smell and the sinuses are apt to become diseased. Aural complications may occur as a result of disturbance of the pressure in the nasopharynx. Although the operation for the removal of tonsils and adenoids is the most frequent cause of acquired stenosis, it is not unusual to find tonsillar remnants still in the throat.

A variety of treatments, most of which have been unsuccessful, have been used in the past. These have included incision of the scar followed by dilatation, use of the cautery, prosthetic appliances and plastic operations, which have varied from the production of a cleft in the palate to the utilization of grafts of mucous membrane or skin. Probably the best method is that described by Nichols in 1896. He believed that this type of stenosis was remediable by the production first of an epithelialized tract laterally in the scarred tissue to which incisions might be made without a recurrence of adhesions. He produced this tract by inserting a silk braid well into the lateral extent of the region of the scarring, the suture being tied and left in place about two weeks. Then another suture was inserted farther laterally. Ordinary mechanical dilatation was used subsequently. The author used this last-mentioned method on his two cases with excellent results. No mechanical dilatation was used postoperatively.

Moore[20] states that when dyspnea arises from acute inflammatory reactions in the pharynx, they usually result from stenosis in or around the tonsillar aperture. He further states that the abscesses, peritonsillar, retropharyngeal and deep abscess of the tongue contribute to the production of acute stenosis. When dyspnea is produced by such abscesses, the most common secondary abscess contributing to the stenosis is submaxillary abscess.

### THE CRICOPHARYNGEAL FOLD AND PHARYNGEAL DIVERTICULUM.

Negus,[21] in discussing the cricopharyngeal fold, laments that modern textbooks contain no accurate description of the anatomy of the mouth of the esophagus. After reviewing the anatomy and physiology of the cricopharyngeal fold, he discusses painful contractions of the cricopharyngeus due to disordered action of the sphincter. The sphincter may exhibit spasmodic contraction, usually as a reflex response to some irritation to the pharyngeal or esophageal wall. This is obviously a favorite site for impaction of foreign bodies. For their safe removal, relaxation of the sphincter is desirable, if tearing is to be avoided. It is most interesting to note that Negus strongly advocates general anesthesia for removing a pointed or impacted foreign body from this region. The value of direct inspection in cases of dysphagia referred to the cricopharyngeal fold is obvious, as a malignant growth may thus be discovered in its early stages, while cure is still a possibility.

Inco-ordination of the cricopharyngeus may have serious results. If the muscle fibres fail to relax when a bolus of food is forced down, considerable strain will be thrown on the walls of the pharynx. In front lies the larynx, at the sides are the alae of the thyroid cartilage and the fibres of the inferior constrictor, but posteriorly there is, during swallowing, a weak area between the circular and the oblique fibres. The weakness at this spot has been brought about by the evolutionary descent of the larynx in man: it does not exist in lower animals. The result of this deficiency may lead to gradually increasing herniation of the mucous membrane with the production of a pharyngeal diverticulum.

All pulsion diverticula are of pharyngeal origin and all tractional diverticula are located in the esophagus and due

to periesophagitis. According to Shallow,[22] pharyngeal diverticula are relatively common and usually require surgical intervention. These diverticula generally originate in a slit below the cricopharyngeus and just above the esophageal fibres. He concurs with Negus that this is the weak point and states that frequently a branch of the inferior artery perforates it. Other areas where diverticula may be found are between the cricopharyngeus and the inferior constrictor, and also at the upper portion of the inferior constrictor.

The symptoms vary according to the duration and extent of the diverticulum. In the beginning the difficulty is negligible, but when the sac becomes large it lies between the esophagus and the vertebral column so as to almost completely close the esophagus. Dysphagia is the most common symptom, while regurgitation is fairly constant and annoying. Other minor complaints are discussed. Diagnosis can always be confirmed by Roentgenographic examination.

Extirpation of the sac with the aid of the esophagoscope is recommended for the cure of pharyngeal diverticulum. Watts[23] reports one case so treated and discharged on the tenth day completely symptom-free. The sac should not only be removed but the defect in the pharyngeal wall should be repaired. Successful accomplishment of this procedure should result in the pharynx and the esophagus retaining their normal anatomic relationship. In attempting to ameliorate or cure the condition, the mechanism of production must be borne in mind. Stretching of the sphincter in the early cases is required. While mediastinitis is always a feared complication there seems to be no reason for it. It should not occur more frequently after the one-stage than after the two-stage operation.

### BENIGN TUMORS.

A nasopharyngeal fibroma which was resistant to radium and inaccessible to electrolysis was successfully extirpated by the transantral approach developed by Sewall for the removal of the sphenopalatine ganglion. Nelson[24] was forced to use this approach because he realized that coagulation or galvanism would have been dangerous owing to the proximity of the major arterial branches. The transantral approach gave excellent access to the pterygopalatine fossa, making possible

the execution there of surgical procedures with complete observance of surgical principles, including good visibility, ease of identification of structures and landmarks, hemostasis by ligature and avoidance of all deformity or loss of function.

Chiappe[25] reports an unusually large fibrolipoma of the hypopharynx occurring in a man, age 68 years. The tumor weighed 20 gm. Nasopharyngeal fibromas on account of their vascularity have earned wholesome respect of surgeons. Many such tumors offer an irresistible temptation to the surgeon for total removal with the snare. The mortality and the grief have been great when this temptation has been yielded to.

A girl, age 17 years, presented a history of an acute coryza followed by nasal obstruction and some interference of speech. There was no pain, no hemorrhage and no ear symptoms. The soft palate was bulged forward by the presence of a large bluish-red tumor which apparently filled the nasopharynx. Doyle[26] reports that under general anesthesia, inspection and palpation showed this tumor to be attached to the basilar process of the occipital bone. It was removed by passing the loop of a tonsil snare through the nose into the nasopharynx and slipping it over the tumor by manipulation with the finger. Practically no bleeding followed the removal. The pathological report showed a pure fibroma with a number of large thin-walled vessels. The most dependent portion of the tumor showed some edema and a small area of myxomatous degeneration. Electrolysis, bipolar coagulation and irradiation are other favorite methods of dealing with such tumors.

The importance of radiography in the study of nasopharyngeal tumors is stressed by Baclesse.[27] He advocates the use of two exposures, a lateral and a mentovertex. The technique is described in detail. If it is necessary, other radiographs may be taken or a tomographic technique may be used.

Clement[28] is of the opinion that adenoidectomy in very young children is an operation full of risks. There are also certain conditions that do not permit surgical intervention in older children. He only mentions tuberculosis as a contraindication to surgery. For all of the cases in which surgery is contraindicated, Roentgen therapy is the method of treatment which has been followed by the most encouraging results. He

states emphatically that this method is without danger and gives the dosage to use in these cases.

### MALIGNANT TUMORS.

Furstenberg[29] frankly acknowledges that we know little or nothing concerning the etiology of malignant neoplasms of the nasopharynx. He says that, whereas we may theorize on the causes of carcinoma of the lip, the larynx and the skin, we are entirely devoid of an explanation for the growth of a malignant tumor high within the vault of the nasopharynx. It is a noteworthy fact that these neoplasms show a striking tendency to infiltrate upward, erode bone and eventually disturb those structures which pass through the basal foramina. Lymphatic metastases to the nodes of the posterior cervical chain occur early and seldom does one see such a lesion creeping downward along the lateral wall of the pharynx; and only in the very late stages of the disease is there sufficient forward extension to produce symptoms referable to the nose.

Malignant neoplasms of the nasopharynx may be classified into four groups, depending upon their tissue derivations and mode of origin.

In the first group are the carcinomas developing from the epithelial structures of the nasopharynx; in the second are the sarcomas taking origin from the connective tissue elements; the third includes the lymphoblastomas arising from the lymphatic structures; and last are the teratological tumors which may undergo malignant degeneration.

The vast majority of malignant tumors of the nasopharynx encountered in the nasopharynx are epithelial in origin and fall into the group of squamous-celled carcinoma. Furstenberg agrees that malignant disease of the nasopharynx presents a most provoking clinical problem and one with a prognosis as futile as that of cancer in any field within the domain of the otolaryngologist. The mortality in typical cases of squamous-cell carcinoma of the nasopharynx is 100 per cent and no therapeutic measures employed by him seem to satisfactorily combat the destructive influences of these lesions.

These tumors show a predilection for the lymphatics about the Eustachian tube and find in this pathway an easy approach to the base of the skull. It must be stated that there is no def-

inite group of symptoms pathognomonic of early pharyngeal carcinoma. Baker[30] comments on the extremely wide variety of symptoms. In the nasopharyngeal group arising near the Eustachian tube, symptoms of deafness, otalgia and tinnitus dominate the picture. Headache is present in all cases, and pain along the distribution of the Vth nerve occurs in some. Sore throat and painful swallowing were the outstanding complaints of the group of oropharyngeal cases, while hoarseness and dysphagia were the predominant symptoms in the laryngopharyngeal group.

## PATHOLOGY OF TUMORS.

The occurrence of congenital tumors in the nasopharynx has been known for a long time and the structures of these pathological curiosities present bizarre findings. Such a case is reported by Howarth,[31] who found a teratoid tumor completely filling the nasopharynx in a baby, age 3 months. The tumor appeared smooth, elastic and was attached to the base of the skull by a broad pedicle. In the discussion of this tumor he quotes Ewing, who has classified these congenital tumors into four groups: dermoids, teratoids, true teratomas and epignathi. Their classification was dependent upon the type of tissue contained.

Because of the belief that carcinoma of the nasopharynx is a relatively common disease, Hauser and Brownell[15] were prompted to review a group of symptoms and signs which often lead to a correct diagnosis in malignant diseases in this region.

A majority of the patients had noted cervical swelling or had symptoms referred to one ear or pain in the ear, while a few had nosebleed, nasal obstruction or sore throat. Seven complained of double vision, and five had a noted unilateral loss of vision. Pain and numbness of the face was the complaint of several more, and two patients had a drooping of one lid, while two more had a noted facial paralysis. Symptoms referable to the nasopharynx may be entirely absent. The tumor, arising in the fossa of Rosenmüller or in the vault of the nasopharynx, may have attention called to its presence not because of its local existence but rather as a result of extension to adjacent tissues or to metastases. Cervical metastasis occurs early in the disease, undoubtedly because of the

highly anaplastic nature of the neoplasm. The duration of symptoms before the correct diagnosis was made varied from the time when the neoplasm was found on routine otolaryngological examination up to five years. In the average case, a little over one year elapsed from the outset of symptoms until the final diagnosis was made.

McGibbon[32] reported two cases of pharyngeal carcinoma and dysphagia with anemia occurring in three generations in one family.

A. J. Wagers,[33] in discussing malignancies of the nasopharynx, quotes Shapiro to the effect that "all patients complaining of pain in the head, face and neck and showing nerve lesions should have the nose and throat examined and the glands of the neck carefully investigated." There is a close relationship between the II, III, IV, the second and third division of the V and the VI cranial nerves.

In a series of 50 cases of malignant neoplasms of the nasopharynx studied by Hauser and Brownell,[15] carcinoma was the diagnosis in 46, three were called lymphoblastoma, and one was diagnosed myeloma. A rhabdomyoma was found in 122 cases of such tumors by Cappell.[34] This tumor is uncommon in any situation. While the histological examination is the only positive manner of making a diagnosis, the naked eye appearance of rhabdomyoma is important. It occurs chiefly in childhood. At first it has the appearance of any simple tumor, but later it presents a polypoid structure which, while it looks soft, is firm to the touch. The lobulated processes are often more translucent and broader at the free than at the attached borders and thus present a typically clubbed appearance.

Hans Brunner[35] reports the interesting case of a man, age 46 years, seeking medical aid because of a sensation of a foreign body in his pharynx while swallowing. He had suffered no pain or dyspnea. Examination disclosed a lesion on the posterior-superior wall of the hypopharynx and biopsy revealed it to be a melanosarcoma. This is a very unusual primary location for such a type of tumor, and although a thorough search was made for an origin elsewhere, it was impossible, even at autopsy, to find any. To add to the interest of this case, another tumor developed from the posterior surface of the epiglottis and could not be accounted for by

contiguity or implantation. It seemed best explained by a personal predisposition for malignancy of the hypopharynx. Several different microscopical diagnoses were made on this second growth before its true nature was established as a melanosarcoma. The only metastasis was to the regional lymph nodes. This patient was treated for a period of approximately four years, the therapy consisting of surgery, radium needles and deep X-ray. An uncontrollable hemorrhage from one of the great vessels of the neck produced death.

Lymphoepithelioma has been the subject of more than ordinary interest and not a little confusion. While there are some who consider such tumors as highly anaplastic carcinomas, transitional-cell carcinomas or lymphosarcomas, many investigators insist that they possess a definite and characteristic histology and, therefore, constitute a separate pathological entity.

Black,[36] referring to the original and independent works of Regaud and Schmincke, expresses their views regarding lymphoepithelioma. They claim that these tumors, besides the above-mentioned distinctive histology, have a certain clinical typicality and an extreme radiosensitivity.

Essentially, the histological feature is a close and constant association of a primitive epithelium and lymphocytes. The explanation for this structure may be found in the normal lymphoepithelium in pharyngeal lymph collections. Out of the welter of confusion regarding the nature of such tumors come several possibilities. They are purely epitheliomatous, with a characteristic of attracting lymphocytes; they are epitheliosarcomas, with the lymphocytes considered as neoplastic cells, or they are reticulum-celled sarcomas with the whole structure arising from the primitive mesenchymal cell.

Cappell[34] thinks lymphoepitheliomas are not all identical. They may be: *1.* the frankly carcinomatous type (Regaud-Jovin); *2.* the round-cell sarcomatous type (Schmincke); or *3.* the transitional-cell carcinomatous type, which differs from the other two in the less intimate association of lymphocytes and malignant epithelial cells.

Hauser and Brownell[15] do not agree with the terms lymphoepithelioma and transitional-cell carcinoma and claim that, according to all basic conceptions of the origin of malignant

neoplasms, an epithelioma can come only from epithelial tissue. So-called lymphoepithelioma and transitional-cell carcinoma are considered by them to be highly anaplastic carcinomas and they are unusually radiosensitive because of their structure.

Those who regard the lymphoepitheliomas as a clinical entity generally agree that their clinical characteristics are: early metastasis to the cervical nodes — even while the primary lesion is small and obscure; a tendency to metastasize to the vertebral column and abdominal organs; and an extreme radiosensitivity. Treatment by any method other than radiotherapy is unavailing.

The IXth, Xth and XIth nerves are also closely related. Vernit's triad indicative of a complete loss of function of the IXth, Xth and XIth nerves consists of: *1.* nasal regurgitation of fluids, due to paralysis of the palate; *2.* dysphagia of solids, due to paralysis of the pharynx; and *3.* hoarseness, due to paralysis of the larynx.

If one is confronted with cervical enlargement and involvement of the cranial nerves at the base of the brain, a full-fledged syndrome of nasopharyngeal malignancy is present and the diagnosis is rather simple. If no irritation of the nerves or loss of function is present, the diagnosis becomes increasingly difficult.

Hamblen and Thomas[37] are of the opinion that whenever possible surgical extirpation is the method of choice in all malignant lesions of the pharynx and larynx; however, in those cases in which surgical intervention is denied, the patient should receive irradiation, and while under treatment he should be hospitalized. Radium has its special uses, and Roentgen therapy is particularly indicated in certain cases. Better results have been obtained with radium needles than with radon seed. Needles are particularly indicated in carcinoma of the tonsil, soft palate and intrinsic carcinoma of the larynx. X-ray therapy is considered the best treatment for rapidly growing carcinomas, and the only treatment when there has been a serious involvement of the lymph nodes.

It has long been the desire of the radiotherapist to obtain a better means of approach to the nasopharynx for purposes of irradiation. In an effort to hold the source of radiation

very close to the tumor, Blady[38] uses a semiflexible shaft, cushioned with a rubber catheter, along the floor of one nasal cavity. A radium capsule is attached by a hinge to the nasopharyngeal end of the shaft. The instrument is introduced and removed in the same manner as a postnasal pack. The capsule is drawn into the nasopharynx in any desired position against the tumor. It is recommended that the radium be left in place for four days. If after five to seven days the mucositis is not too severe, another intracavitary treatment is instituted. The dosage is the same.

Leroux[39] is of the opinion that in the treatment of malignant conditions of the nasopharynx, surgical diathermy should be used in combination with radiotherapy and radium. He reports excellent results in those cases in which this particular combination was used. In certain types of cases, diathermy is to be preferred to surgical measures which so frequently are attended with serious postoperative complications.

Hayes E. Martin[40] reminds us that in the region of the pharynx are found practically all the lymphoepitheliomas and transitional-cell carcinomas that are characteristic of the upper parts of the respiratory and alimentary tracts. They are highly malignant, metastasize early and are comparatively radiosensitive. He believes that treatment is mainly a problem of radiation. While certain anatomical structures permit of surgical removal, it is seldom that cancer in these regions is discovered before it has involved adjacent inoperable areas. Hauser and Brownell[15] express the opinion that the treatment of malignancies of the nasopharynx is very unsatisfactory, and Furstenberg[29] states that the mortality in typical cases of squamous-cell carcinoma is 100 per cent. Orton[41] believes that treatment and prognosis depend to a great extent upon whether or not the starting point of the lesion is known. Martin[40] admits that radiation therapy was not successful prior to the fractional dose principle as developed by Coutard. The pharyngeal cavity is unsatisfactory for the general use of interstitial irradiation, and the accompanying edema or radionecrosis in hypopharyngeal lesions are of great importance. Experience has shown that exclusive use of Roentgen therapy in all cases is not the best solution of the problem. Supplementary irradiation with radon implants in combination with a surgical approach is the best. Robert C. Martin[42] informs

us that the action of radium, screened so that the gamma rays alone are effective, is similar to that of the Roentgen rays; therefore, cells undergoing mitotic division and those rich in nuclear chromatin are more responsive to radiotherapy. If the tumor responds, there is either autolytic degeneration of the cells, which is the desired result, or an arrest of the tumor cells so that surgical removal may have a better chance of success. The tumor bed undergoes inflammatory reaction, ending in a fibrosis and an obliterative endarteritis, which materially reduces the blood supply to the tumor. Infection is a contraindication to the use of radiotherapy as the resistance of the tissue is impaired and severe sloughing may result. The immediate effect of radiotherapy is an epithelitis with erythema and small patches of fibrin on the mucosa, as well as some swelling.

Justras[43] reported his observation on a series of 48 cases of lymphosarcoma treated by Roentgen rays. Sixteen of these were five-year cures. In 40 of the cases the point of origin was as follows: 24 from the palatine tonsil, 13 from the pharyngeal and paratubal tonsils, and three from the lymphatic tissue at the base of the tongue. These lesions occurred almost equally in the two sexes. Eighty-three per cent of the patients were over 40 years of age, and the best end-results were obtained between the ages of 40 and 60 years. This author concurs with Hays Martin in advocating the use of fractional irradiation. It is contended by some observers that since pharyngeal cancer metastasizes early and widely in the neck, the entire potentially node-bearing area should be included in the skin portals so that all early and palpable metastases will be treated. Theoretically this is sound, but it is not practical because the upper limit of safe dosage with large portals is insufficient to destroy moderately radioresistant tumors capable of control by higher dosage with smaller portals. Up to a certain point, the effect of irradiation by any one method of irradiation increases in proportion to the dose, and beyond this point the benefits not only do not decrease but harmful results are obtained. Since all of the harmful results of external and interstitial irradiation are not identical, a combination of the two when possible is desirable. The general feeling of the radiotherapist is that the tumor must be destroyed in the first series of radiations. If insufficient therapy is given initially, the tumor fails to respond well to

subsequent therapy; if the tumor is of the radioresistant type, it becomes more so by virtue of the initial series. All patients receiving heavy doses of radiation about the pharynx complain of an annoying sensation of dryness, which usually lasts about a year. Orton[41] points out that treatment of the posterior pharyngeal malignancies gives good results only if the patient is seen early. Growths in the pyriform sinus, epipharyngeal and hypopharyngeal areas have the poorest prognosis, although if they are seen at a very early stage, surgical intervention may be valuable. In this connection, he discusses the main features of Trotter's operation, the lateral transhyoid pharyngotomy, as a surgical approach to the pharynx.

## COMPLICATIONS.

Complications arising from nasopharyngeal tumors will depend primarily upon the exact location and extent of the growth. It is obvious, says Charles L. Martin,[44] that when a tumor of the nasopharynx extends into the adjoining soft tissues, the Eustachian tube soon becomes blocked, and deafness, tinnitus, a feeling of stuffiness in the ears, retraction of the drum and otitis media usually result. The anterior pillar of the affected side is pushed forward and eventually the soft palate may be invaded. Later the pterygoid and masseter are attacked and the victim can then open the mouth very little, if at all. In about one-third of the patients afflicted with pharyngeal carcinoma, the sphenoid bone is invaded. Involvement of practically all of the cranial nerves have been reported. Martin further states that the first lymph nodes to enlarge are felt high up in the neck behind the angle of the jaw, and a little later the submental group may become palpable. The adenopathy increases rapidly, is often accompanied by pain and a wry neck, and produces the clinical picture of Hodgkin's disease, for which it is frequently mistaken.

Hayes E. Martin,[40] in discussing the general systemic reactions from irradiation, informs us that chronic radiation sickness is of frequent occurrence. The patient is listless, weak and toxic, and becomes cachectic. This is due to partial derangement of glandular, vascular and nervous function of the neck, and is probably not the result of local discomfort or lack of nutrition from dysphagia. This can be prevented by limiting the radiation dosage. The acute effect in the skin and

mucous membrane is usually a bit alarming at first. There is a blistering of the skin, and while it appears serious it always heals if properly cared for. The mucosal reaction proceeds to a membranous mucositis of diphtheritic appearance. While not painful at rest, it is painful to motion and bleeds freely. During the height of reaction there is diminution in amount and increase in viscosity of saliva. Irrigations of warm sodium bicarbonate help dysphagia, and dyspnea is always present when reaction is below level of soft palate. In discussing the treatment of cervical mestastasis, Hayes Martin[40] concurs in the general belief that these glands as a rule are unsuited to neck dissection because of their high degree of malignancy, rapid growth, frequent bilateral distribution and inaccessibility. When possible, these metastatic enlargements should be irradiated at the same time as the primary lesion, and usually they are found to be more resistant than the original tumor.

Radionecrosis when not too extensive is not a very serious complication except in the hypopharynx. It is especially likely to occur where supplementary interstitial irradiation is used. Hemorrhage may be due to deep radionecrosis. Definite disturbances of thyroid function during acute reaction following radiation occasionally occurs, sometimes necessitating thyroid therapy.

Probably one of the most annoying of complications following radiations is xerostomia. This chronic drying of the mucous membranes of the mouth is due not only to the disturbance of function of the major salivary glands but also to a disturbance of minor salivary glands in mucosa of palate, pharyngeal walls and base of tongue. If relief is not obtained by local applications, assurance may be given of complete relief in due course of time.

## BIBLIOGRAPHY.

1. Worth, E., and Braun, T.: Comparative Bacteriology of the Epipharynx, the Nose and the Tonsils, and Acute Infections. *Arch. Ohren., Nasen.. u. Kehlkoph.*, 141:317, 1938.

2. Burghi, S. E.: Simple Acute Rhinopharyngitis of Childhood. *Arch. Pediat.*, Uruguay, 9:653-698, Nov., 1938.

3. Schenck, H. P.: Chronic Infection. *Pa. Med. Jour.*, 41:578, April, 1938.

4. O'Conner, T. P.: Low Grade Fever, Results of Infection. The Laryngoscope, 48:38-50, 1938.

5. Bajkay, T.: Adenoid Vegetations in Elderly Persons. *Arch. Ohrenrheilk u. Heilk.*, 145:92-94, July, 1938.

6. King, J. I.: Pharyngeal Infection in Exophthalmic Goitre. *Bull. Johns Hopkins*, 62:593-609, June, 1938.

7. Banyai, A. J.: Tuberculosis — Cod Liver Oil for Local Treatment. *Arch. Otolaryngol.*, 27:154-160, Feb., 1938.

8. Richardson, J. R.: Nasal Airways. *Rhode Island Med. Jour.*, 21:149-152, Oct., 1938.

9. Laurent, L. J. M.: Hemophilia with Fatal Spontaneous Hemorrhage from Pharyngeal Abscess. *Brit. Jour. Child. Dis.*, 34:282-286, Dec., 1937.

10. Gerocarni, A.: Rare Case of Retropharyngeal Abscess in an Adult. *Boll. Mal. Orechio.*, 57:53-60, Feb., 1938.

11. Liebermeister, G.: Retropharyngeal Abscess. *Deut. med Wchnschr.*, 64:337-338, March, 1938.

12. Merklin, L.: Retropharyngeal Abscess with Fistulization Into the External Auditory Canal; Report of Three Cases. *Arch. Pediat.*, 55:395-399, July, 1938.

13. Kecskes, Z.: Pathology and Therapy of Pre-Epiglottic Phelgmone. *Monatsschr. Ohrenheilk.*, 72:387-392, April, 1938.

14. Ciurlo, L.: New Method of Rhinopharyngeal Examination. *Valsalva*, 14:434-438, Sept., 1938.

15. Hauser and Brownell: Malignant Neoplasms of the Nasopharynx. *Jour. A. M. A.*, 27:2467-2472, Dec., 1938.

16. Abramowicz, Louis: Dysphagia Caused by an Anomaly of the Styloid Process. *Les Ann. d'otolaryngol.*, May, 1938.

17. Johnson, L.: Certain Considerations on Dysphagia Associated with Anemia. *Ann. Otol., Rhinol. and Laryngol.*, 47:809-813, Sept., 1938.

18. Childrey, John R.: Stenosis of the Nasopharynx. Two Case Reports. *Ann. Otol., Rhinol. and Laryngol.*, 46:1118-1123, Dec., 1937.

19. Borghesan, E.: Operative Technique of the Osseous Choanal Diaphragm. *Valsalva*, 14:73-75, Feb., 1938.

20. Moore, R. S.: Acute Stenosis of Pharynx. *N. Y. State Jour. Med.*, 38:434, March, 1938.

21. Negus, V. E.: Affections of the Cricopharyngeal Fold. The Laryngoscope, 48-857-858, Dec., 1938.

22. Shallow, Thos. A.: Pharyngeal Diverticulum. *Jour. Internat. Col. Surg.*, 2:48-52, Jan., 1939.

23. Watt, J. L.: Pulsion Diverticulum of the Pharynx. *Med. Jour. Australia*, 2:905-907, Nov., 1938.

24. Nelson, Roy F.: Nasopharyngeal Fibroma. *Arch. Otolaryngol.*, 27:313-317, March, 1938.

25: Chiappi: Voluminous Fibrolipoma of the Hypopharynx. *Valsalva*, 14:449-450, Sept., 1938.

26. Doyle, George F.: Report of a Case of Telangiectatic Edematous Myxofibroma of the Nasopharynx. *Ky. Med. Jour.*, 36:421-423, Oct., 1938.

27. BACLESSE, F.: Radiological Study of the Normal Rhinopharynx. *Arch. Elec. Med.*, 46:161-178, July, 1938.

28. CLEMENT, R.: Roentgen Therapy of Adenoiditis. *Bull. Soc. Pediat.*, Paris, 36:473-480, July, 1938..

29. FURSTENBERG, A. C.: Cancer Nasopharynx. *Surg., Gynec. and Obstet.*, 66:400-404, Feb., 1938.

30. BAKER, D. C.: Pharyngeal Cancer. THE LARYNGOSCOPE, 47:904-908, Dec., 1937.

31. HOWARTH, WALTER: Teratoid Tumor of the Nasopharynx. *Jour. Laryngol. and Otol.*, 53:386-387, May, 1938.

32. MCGIBBON, J. E. G.: Pharyngeal Carcinoma and Dysphagia with Anemia. *Laryngol. and Otol.*, 53:32-34, Jan., 1938.

33. WAGERS, A. J.: Sarcoma of the Nasopharynx with Report of One Case. THE LARYNGOSCOPE, 48:302-304, May, 1938.

34. CAPPELL, D. F.: Pathology of Nasopharyngeal Tumors. *Jour. Laryngol. and Otol.*, 53:558-559, Sept., 1938.

35. BRUNNER, HANS: A Peculiar Case of Melanosarcoma of the Pharynx. *Jour. Laryngol. and Otol.*, 53:509-518, Aug., 1938.

36. BLACK, J. I. MUNRO: The Lymphoepitheliomata. *Jour. Laryngol. and Otol.*, 53:225-245, April, 1938.

37. HAMBLEN, THOMAS C.: Treatment of Carcinoma of Pharynx and Larynx by Irradiation. *Brit. Med. Jour.*, 2:447-448, Aug., 1938.

38. BLADY, JOHN V.: A New Instrument for Irradiation of Tumors of the Nasopharynx with Radium or Radon. *Amer. Jour. Roentgen.*, Vol. 40, 1938.

39. LEROUX, L.: Surgical Diathermy in Malignant Tumors of the Pharynx. *Oto-rhino-laryngol. Internat., Lyon*, 26:11-15, Jan., 1938.

40. MARTIN, HAYES E.: Treatment of Pharyngeal Cancer. *Arch. Otolaryngol.*, 27:661-691, June, 1938.

41. ORTON, HENRY L.: Cancer of Laryngopharynx. *Arch. Otolaryngol.*, 28:344-354, Sept., 1938.

42. MARTIN, ROBERT C.: Malignancies of the Nose and Throat. *Ann. Otol., Rhinol. and Laryngol.*, 47:512-521, 1938.

43. JUSTRAS, A.: Protracted Roentgen Therapy of Pharyngeal Lymphosarcoma. *Amer. Jour. Roentgenol.*, 39:792-798, May, 1938.

44. MARTIN, CHARLES, L.: Complications Produced by Malignant Tumors of the Nasopharynx. *Amer. Jour. Roentgen.*, 41:3, March, 1938.

632 Maison Blanche Building.

# STUDIES IN LABYRINTHINE FENESTRATION TO IMPROVE HEARING (A PRELIMINARY REPORT).

DR. SAMUEL J. KOPETZKY, New York.

The surgery for the relief of deafness has been given trial in this country, and both operative work and experimental studies on various phases of the problems concerned are finding publication in our literature.

For many years the problems which concerned the deafened interested me. In October, 1937, when Sourdille presented his operative therapy for progressive deafness and otosclerosis, I[7] opened the formal discussion with a plea that his technique "was feasible," and I asked for "an avoidance of skepticism" when studying his surgical methods. When I had studied the prospects which surgical therapy promised in selected cases, I drew the attention of American otology in May, 1938, to the possibilities and the probabilities of achieving beneficial results in cases of deafness by surgical therapy; and *at that time* I particularly and specifically directed attention to the Lempert operation,[1] and advocated that it be given a trial. I hoped that further study of this technique and continued observations of the results obtained from its employment would eventually give it its proper place in our surgical therapy.

I am, however, less interested in any particular surgical technique than I am in the many-sided problems which surgical relief for the deafened embrace. In the long run, the question of adopting a specific surgical technique will settle itself by the "trial and error" method.

One may divide all reports on surgical therapy for the deafened into three phases. The first, the occasional, casual and cautious attempts which are scattered in the literature. The second is the empirical application of surgical measures. Here I place Sourdille's[2] operative reports, and the reports found in our own literature. The third is the study of the problems which this surgery brings in its train, and to the

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Oct. 7, 1939.

solution of which only the very beginnings have been made. In fact, most of the problems have not, as yet, been clearly defined or even enunciated. A commencement has been made by Holmgren,[3] who seeks the establishment of fundamental factors upon which to predicate a rational surgical therapy. His studies of various means to keep the labyrinthine fistula open, and Nager's[4] histological studies are the first efforts to take fistulization out of the realm of empiricism and into one of rationalism based upon adequately observed factual data. Canfield,[5] too, is engaged in finding some rational basis for this type of surgery. To this date that is all that otological literature reveals along this line. What Canfield promises to undertake should clarify many perplexing questions which reports on operated cases cannot settle.

My own work is not yet ready for final report. I will, however, take this opportunity to present some "sample cases" from my material, to draw attention to some characteristic features — some of which definitely substantiate the report recently made by Campbell.[6] In addition, I present other data which gives its own arguments in contrast to his.

If the effort to acquire the necessary technical dexterity to perform this type of surgery leads to the dissecting room, I hope my contribution will begin a drive to the physiological laboratory, because the whole question of "how we hear," what structures come into play "while hearing" and "while listening" is in need of clarification. Theories of hearing, based upon premises which in themselves are equivocal, need re-examination. They are largely built upon unproved foundations, and the very light which is being shed by the results obtained by our empiric operative procedures may perhaps lead to new conceptions in the fundamentals of the physiology of hearing.

In confirmation of Dr. Campbell's exceptions to standardized procedures, I state that the endaural approach must not be considered the essential approach. There are no objections to its employment by those who desire to use it to expose the temporal bone, but it should be emphasized that all steps in the technique of labyrinthine fenestration can be done equally well through the usual, the open postauricular route, and the same direct vision, the same inter-relationship of parts is equally well obtainable. For those of us who have resident

and staff assistants at operations, who must be trained in otolaryngology, the open, postauricular approach offers obvious advantages.

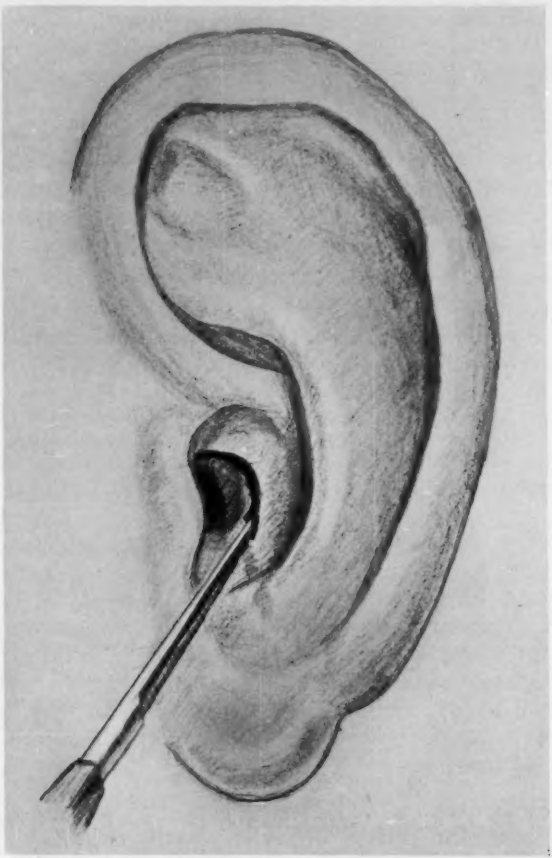


Fig. 1. Incision of superoposterior edge of external auditory canal with concha, to free flap.

In using the postauricular approach, this figure shows the incision to be made as the first step in the technique. Fig. 2, which follows, shows the exposure obtained when the concha

is retracted toward the face, and the mastoidal and epitympanic areas have been exposed.

The kind of membrane which covers the fenestration is not the factor responsible for the permanence of a labyrinthine fistula. This Canfield[5] has shown experimentally, and my own observations on the living patient, as well as Campbell's, sub-

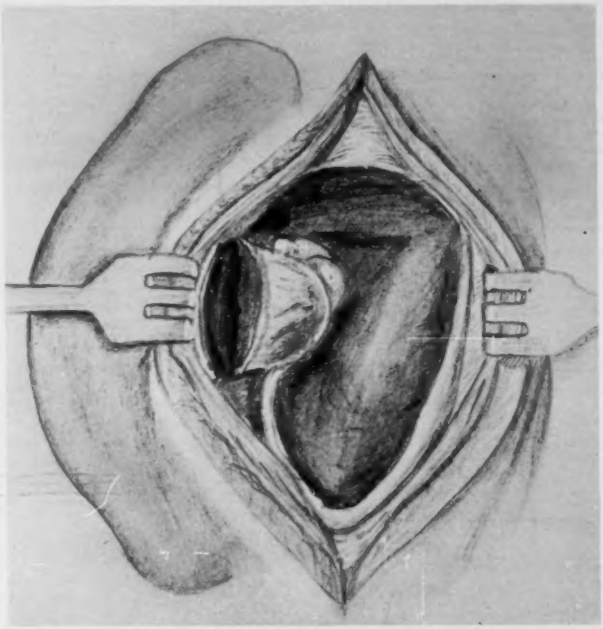


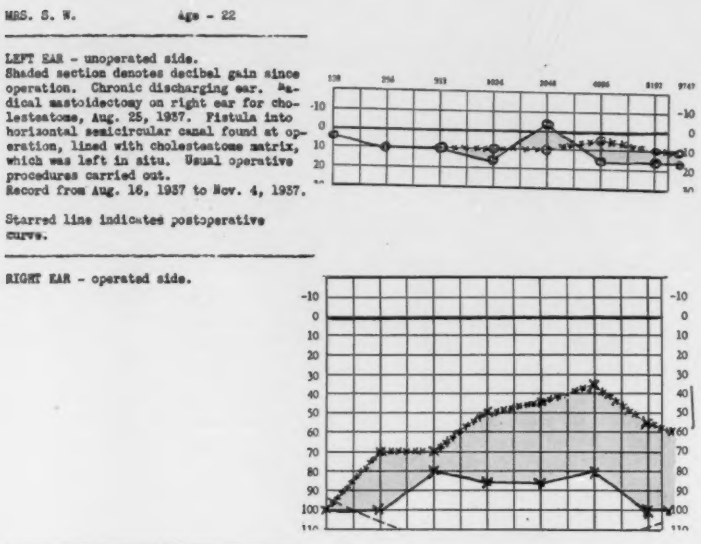
Fig. 2. Exposure obtained by freeing concha, turning it toward the face, showing cut surfaces of external auditory canal wall. Also remaining part of membranous wall, bony wall removed. Epitympanic space exposed. Partly diagrammatic.

stantiate his findings. Nor is the following of an exactly outlined, definite technique the *sine qua non* which determines success or failure. Campbell implies this, too, and I agree with him.

Here I report only sample cases. Only the pertinent factor in each is stressed. The complete case report will be published when more time for observation has elapsed. I, there-

fore, present only such data as will serve to elucidate the observations which I now desire to place on record.

*Case 1:* A female patient with a spontaneous fistula covered with cholesteatomatous matrix. The cholesteatome was removed surgically. There is now an epidermatized radical




RECORD OF CONVERSATIONAL LOSSES

| Date | Right | Left |
|---|---|---|
| Aug., 1937 | 65% | 7% |
| Nov., 1937 | 44% | 8% |

Fig. 3.

cavity. The hearing acuity is definitely improved after operation (see Figs. 3 and 4).

Fig. 3 is a self-explanatory contracted chart of the case. I draw attention to the excellent hearing acuity in the *normal, unoperated* ear, in contrast to what I show later in other types of cases.

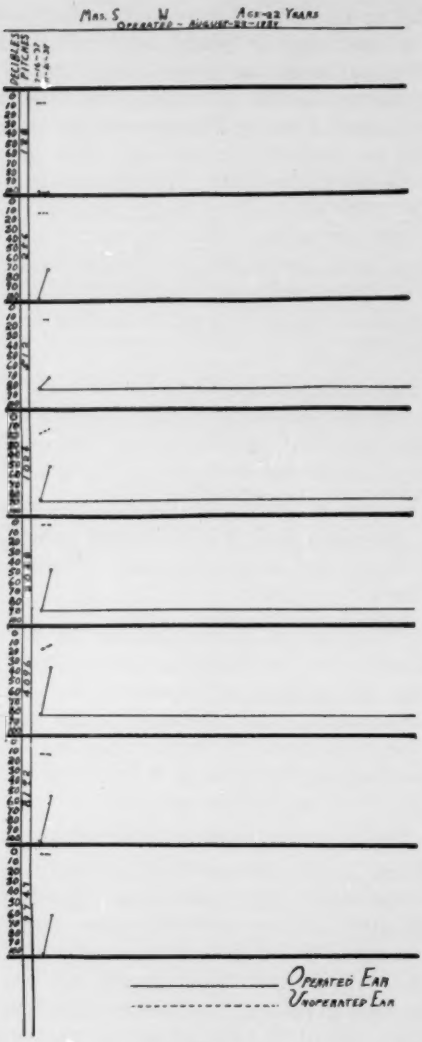



Fig. 4.

FOOTNOTE: Fig. 4, and others which will follow, is a "breakdown" of the several audiometric recordings, so that the record for all tones over the period of observation may be studied separately. Thus, it is possible to record many successive audiometric tests and analyze the trend of the curve for each note.

In contrast to this case, I have a record of a chronic mastoiditis in a man, age 49 years, who had a cholesteatome which eroded his horizontal semicircular canal, and gave a positive fistula test before operation. His hearing acuity in percentages showed a loss of 58⅞ per cent in the right ear, and a loss of 10⅝ per cent in the left ear. The radical mastoidectomy was performed on Nov. 1, 1935, on the right ear. In September of 1939 his hearing acuity showed a 59⅝ per cent loss in the right ear, and a loss of 12½ per cent in the left ear; with a patent, open fistula still present in the external semicircular canal of the right ear, upon which the operation had been performed. I was able to get a positive fistula test. The ear has been dry.

I report this outlined case to make the point that an open fenestration is not the *only factor present* in these cases which brings improved hearing in its train. From what I describe below, additional evidence on this point is presented.

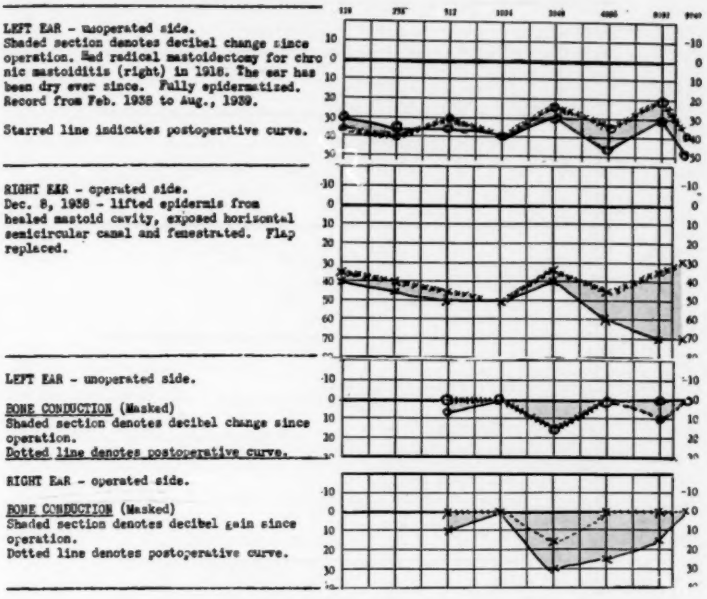
*Case 2* is one in which a healed radical mastoid cavity was taken after 20 years and the horizontal semicircular canal fistularized. Fig. 5 is a contracted chart of this case, which is self-explanatory.

Fig. 6 is a chart of the hearing acuity for all notes on the Western Electric 6A Audiometer, from Dec. 7, 1938, to Aug. 18, 1939. There is an improvement for all tones. There is also an improvement for all tones in the unoperated ear. The fistula remained open and active until May — a period of five months. The hearing remains at this level *even though no fistula test reaction can now be produced.*

*Case 2* is shown to demonstrate that the presence of tympanic structures is not essential to a successful result from a fistulization operation. The opposite ear also improved. The fistula closed after an intercurrent upper respiratory infection. The hearing at this time still remains improved. In these two cases, one with a spontaneous fistula and one with a surgically-made fistula, radical mastoidectomies had been done. Note the improvement in hearing in the unoperated ear for most tones.

*Case 3* is shown in Figs. 7 and 8. The middle ear and the epitympanic space have been left *undisturbed.* The rationale for interfering with these structures has hitherto never been

satisfactorily clarified. To test the advantage or disadvantage of leaving the epitympanic space undisturbed, I first exenterated the mastoid process, extended the procedure to outline sharply the horizontal semicircular canal — which was fenestrated; and then the membranous posterior meatal wall in its




RECORD OF CONVERSATIONAL LOSSES

| Date | Right | Left |
|---|---|---|
| Feb., 1938 | 37% | 28% |
| Aug., 1939 | 54% | 25% |

Fig. 5.

entirety was adapted to cover the fenestrum. The hearing acuity is shown in Figs. 7 and 8.

Hearing for conversational tones has improved 8 per cent in the right ear and 4 per cent in the left. From all standpoints these charts are unimpressive, and one could not call this case a success, despite the fact that *the fistula is still*

*active.* Yet this small gain in hearing acuity satisfied the patient and he is contented. Note that the hearing in the *unoperated* ear has also improved since operation.

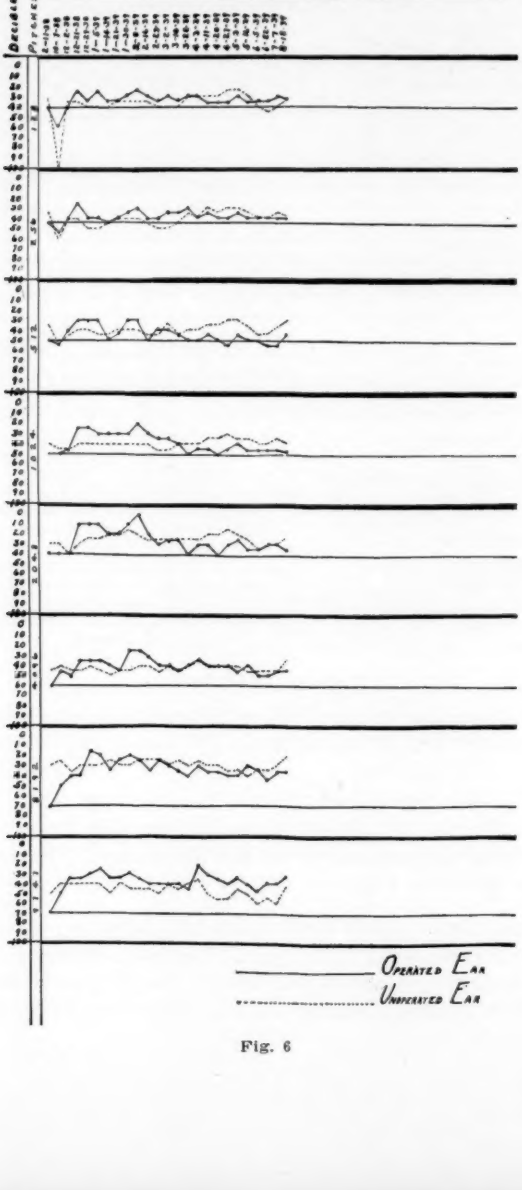
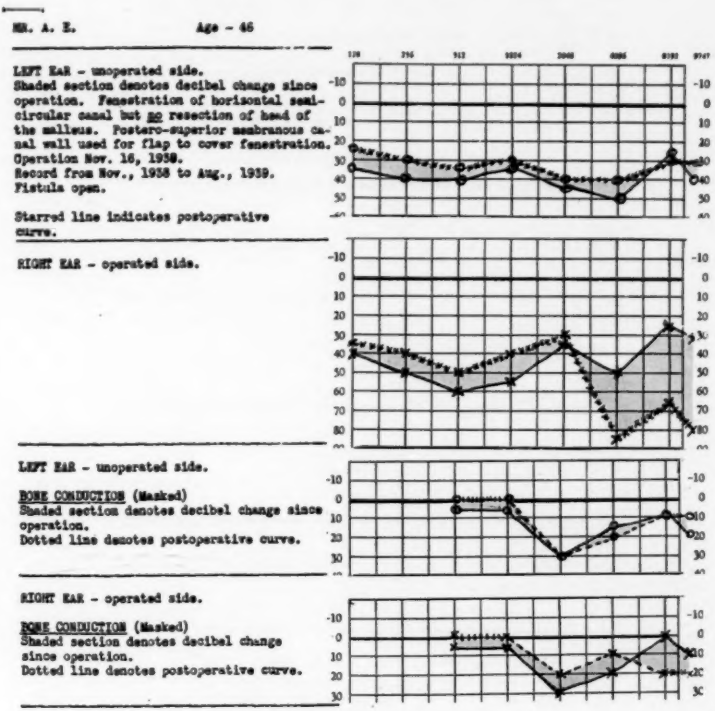



Fig. 6

At this point permit me to stress that while there is no better criterion for estimating hearing acuity than the audiometer, both for accurate recordings and for standards of comparison, it is apparent to anyone who operates on these




RECORD OF CONVERSATIONAL LOSSES

| Date | Right | Left |
|---|---|---|
| Nov., 1938 | 40% | 32% |
| Aug., 1939 | 32% | 20% |

Fig. 7.

cases that much more is accomplished than what the audiometer records. I shall revert to this again later.

In *Case 4*, sketched in Fig. 9, the tympanic and the epitympanic spaces were also left *untouched*. The horizontal semi-

circular canal was fenestrated and covered by the flap from the posterior membranous canal. The chart of the hearing in this case definitely is *not impressive.* In the conversational

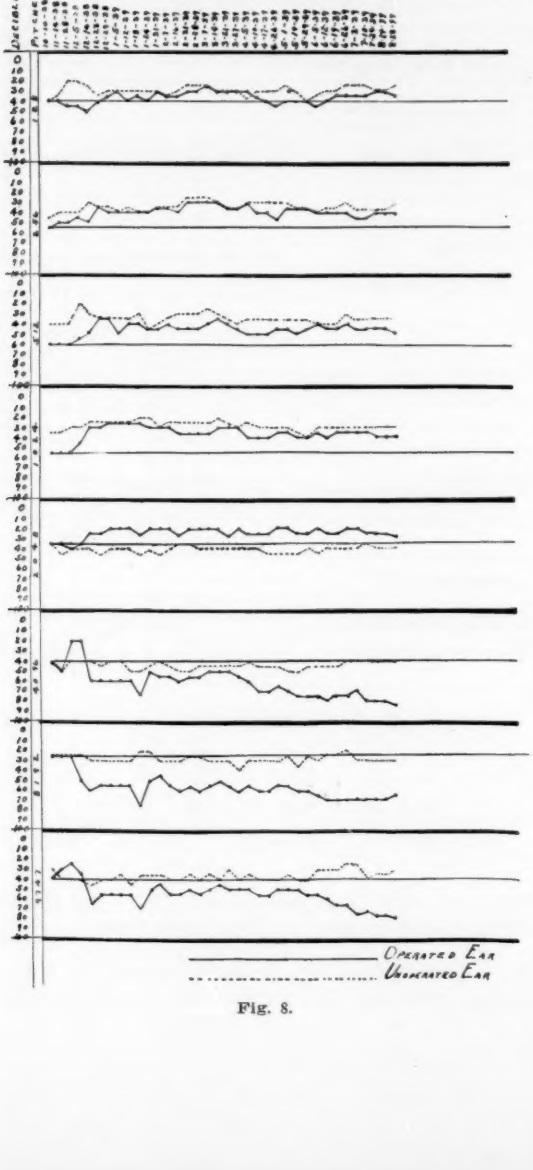
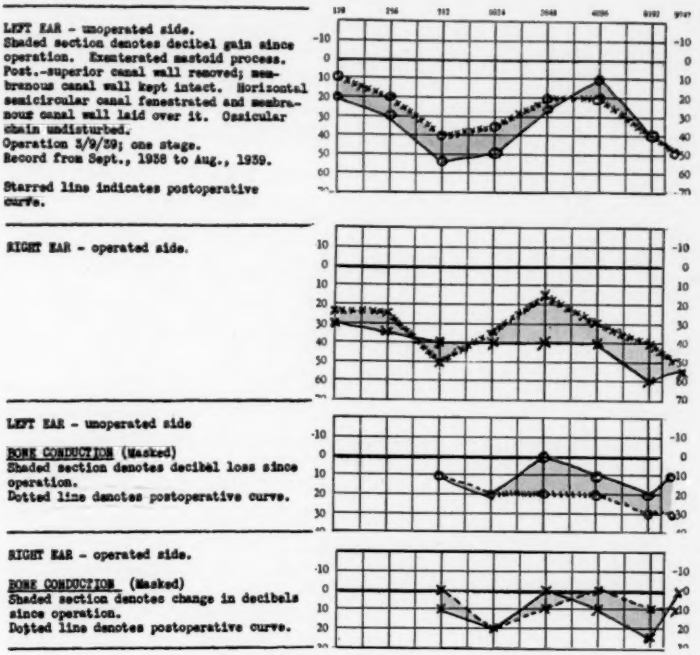



Fig. 8.

range of the 512, 1,024 and 2,048 actaves, there is no marked improvement, even though the fenestration remains open. There is some degree of improvement in hearing, found in the operated ear as well as in the unoperated ear. The parallelism in the curve in both ears is to be noted. In my introductory




RECORD OF CONVERSATIONAL LOSSES

| Date | Right | Left |
|---|---|---|
| Sept., 1938 | 32% | 34% |
| Aug., 1939 | 28% | 24% |

Fig. 9.

remarks I stated that many factors must be considered in estimating the value of fistulization. This patient suffers from a nasal allergy. Every time there is an increase in the intensity of the allergy, there is a drop in the hearing acuity in

both ears. If one studies Fig. 10, the audiometric recordings seem better but, as I remarked before, there is more to hearing than shown on the audiometric charts, or for that matter

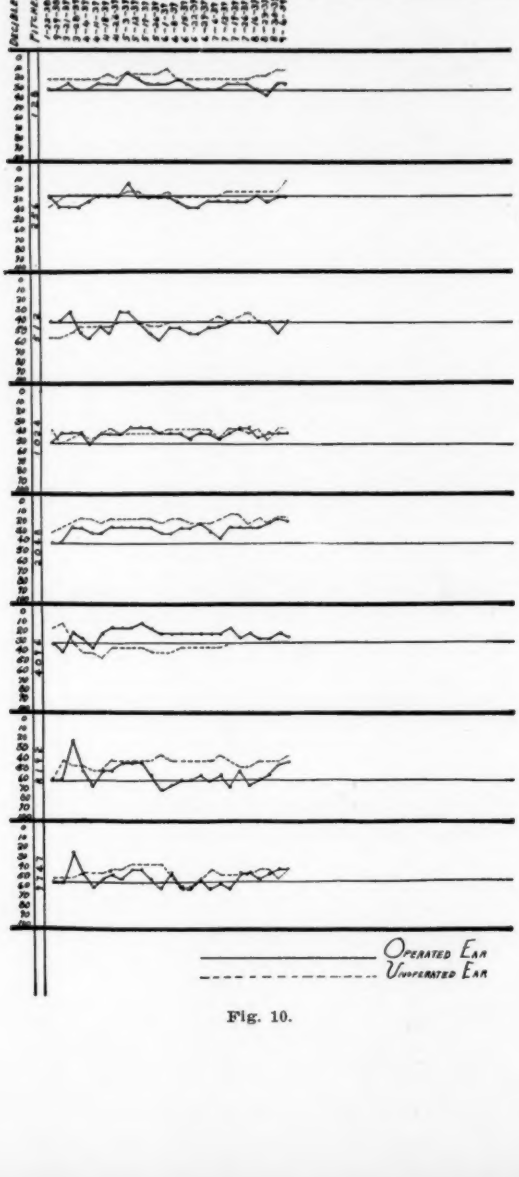



Fig. 10.

in the results of testing with tuning forks. In this case, with an open fenestration yielding a *positive fistula reaction*, hearing becomes worse during the attacks of allergy. Is this not further evidence that a permanently *open fenestrum* in the bony labyrinth capsule *is not the sole factor* in establishing and in maintaining better hearing acuity? I call your attention to the drop in the bone conduction in this case. I cannot yet explain this, since — as I will show further on — such finding is not incompatible with an excellent result for air conduction (see Fig. 16).

I have other cases whose fenestrations still remain open, but which, after an incidental upper respiratory infection, have lost the newly gained hearing acuity acquired by the operation. Campbell makes a similar observation, but in some of my cases I find that the hearing has not regained its increase after the infection subsides, although the fistula has remained open. This, too, needs clarification.

In *Case 5*, shown in Fig. 11, the hearing chart is that of a two-stage operation, based upon the combined technique of Sourdille and Lempert. The first stage consisted in the exenteration of the mastoid process, the removal of the bony external auditory canal wall, the freeing of the drum by removing the sulcus tympanicus, opening into the epitympanic cavity, and separating the articulation of the major ossicles and removing the head of the malleus. The flap was then placed over the *unopened* semicircular canal and the mastoid cavity was permitted to heal and epidermatize. The second stage consisted in elevating the thin epidermal layer over the mastoidal area, exposing the horizontal semicircular canal, fenestrating it and then replacing the dermal flap. The shaded portion of the chart denotes the tests taken during the first stage. This shows a loss in hearing acuity for both the *operated* and the opposite ear. After the second stage was completed, increased hearing in both ears for all tones is clearly evident. The operated ear is much better than the opposite ear. From all standpoints this must be considered a successful outcome thus far.

In viewing Fig. 12, the great increase in the conversational range is striking. This woman, age 45 years, would hardly have been considered to hold out prospects for such a successful outcome. Her bone conduction, with masking, taken prior

to operation for the second stage, would certainly be a contra-indication if we are to accept the presently proposed guides.

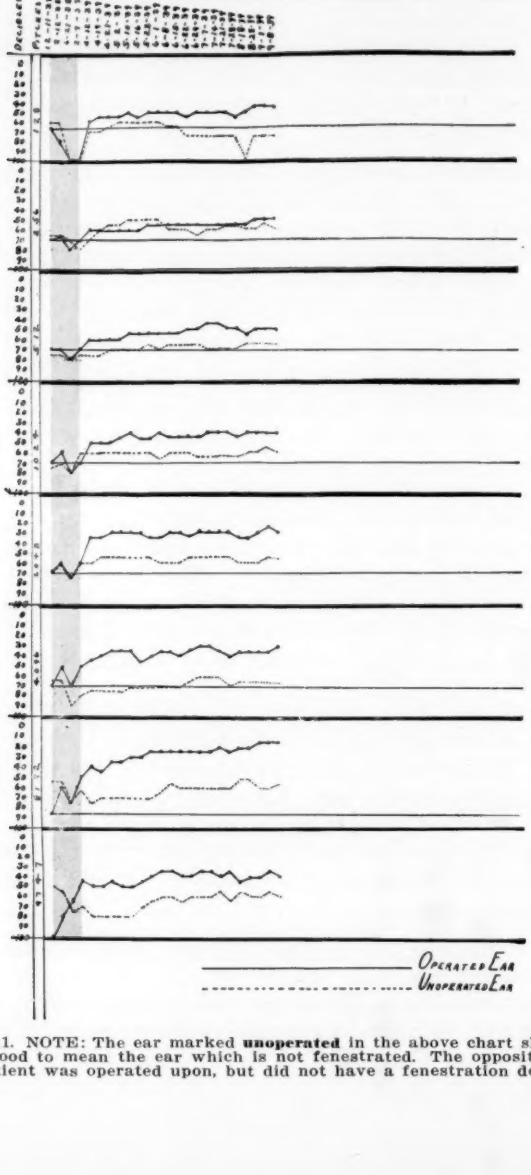
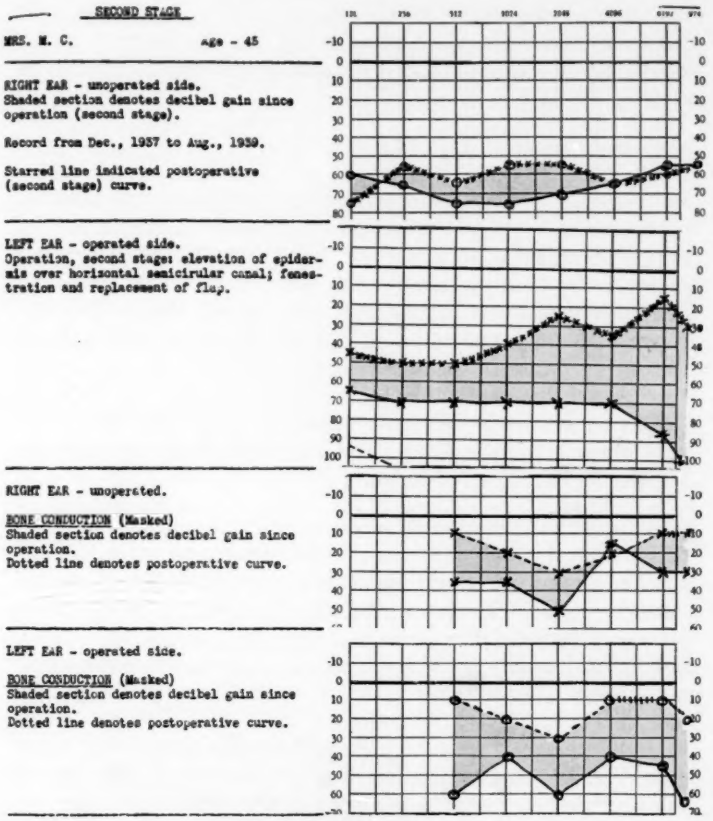



Fig. 11. NOTE: The ear marked **unoperated** in the above chart should be understood to mean the ear which is not fenestrated. The opposite ear in this patient was operated upon, but did not have a fenestration done.

But of greater moment is the fact that the improvement in her air conduction is *here paralleled by an improvement in the*




RECORD OF CONVERSATIONAL LOSSES

| Date | Right | Left |
|---|---|---|
| Dec., 1937 | 56% | 56% |
| Aug., 1939 | 47% | 31% |

Fig. 12.

*bone conduction (taken under conditions of masking)*, and the fistula remains open after a seven months' period of observa-

tion. She not only hears well, but her awareness of her surroundings is better. We have no means of recording this. This case, as well as those shown in Fig. 3 and Figs. 4, 5 and 6, prove conclusively that the permanence of the fistula is not achieved by the tissue which covers it. What is necessary is that the tissue should eventually take on the characteristics of periosteum and, to hasten healing and secure a dry ear, be clothed by pavement epithelium.

*Case 6,* shown in Fig. 13, is the record of a one-stage operation, with resection of the head of the malleus, *maintaining the incus in situ,* and fenestration of the horizontal semicircular canal — using the posterior-superior portions of the membranous canal to cover the fenestrum. Note the gradual increase in hearing acuity for each tone. Note also the improvement in the unoperated ear for all tones. For the conversational range of 512, 1,024 and 2,048 the operated ear shows an impressive improvement; the opposite ear shows marked improvement only in the lower ranges, but little for the 1,024 or the 2,048 notes. There is improvement in the opposite ear for the 4,096 tone, and a marked improvement for the 8,192 and 9,747 tones.

A factor in this case deserves mention, namely — that the flap sloughed off after 15 days. Since that time there has *not been any covering* over the fistula except a blanket of granulations — which I am continually cauterizing with silver nitrate. The fistula has remained actively open for 11 months, and is still open. This case, too, is still under observation and study. Her hearing in the operated ear is now within normal limits. The audiograms and the "breakdown" chart show this. Both ears are improved. For conversational tones (see Fig. 14) the percentage loss is now only 9 per cent in the operated ear, and 15 per cent in the unoperated ear.

Where Campbell reports on the question of flaps, I ask — what about the necessity for a particular type of flap, about which there has been so much stress? This case is no accident. The permanent patency of the fistula here has shattered all my former beliefs concerning the importance of the covering membrane. There are definite factors in the *making* of the fistula which inhibit bone proliferation. When these are achieved, a fistula will remain open and hearing acuity will

be increased in the given case — regardless of what covers the fistula.

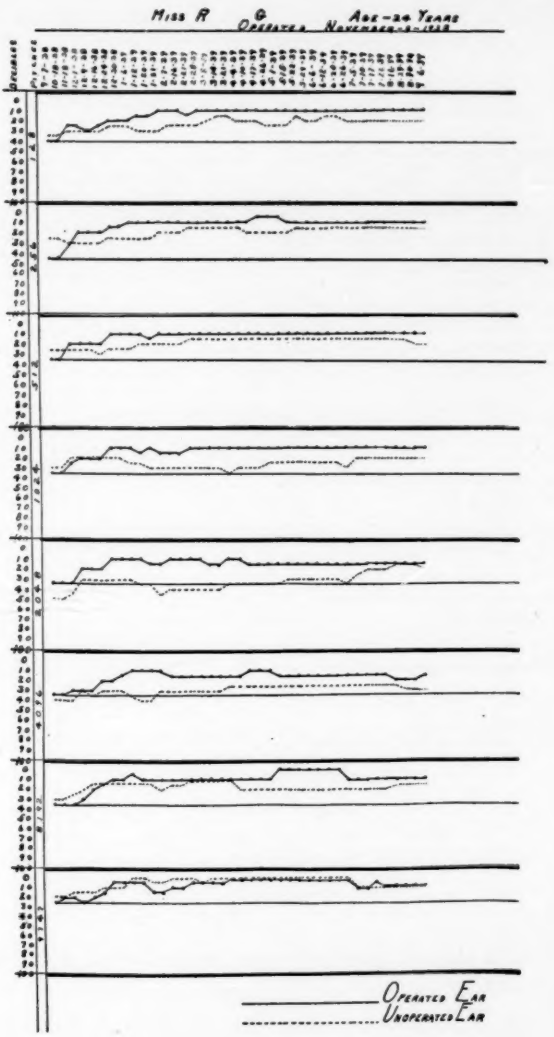
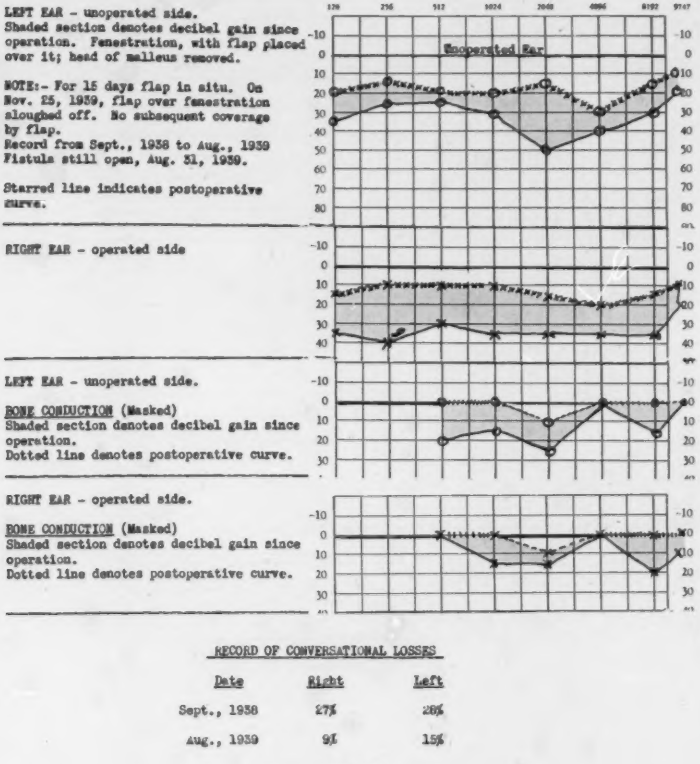



Fig. 13.

*Case 7*, shown in Fig. 15, is a record of the hearing acuity of a case operated on in a one-stage operation, head of the malleus resected, the *incus removed* and the posterior meatal membranous wall used to cover the fenestration. I call your attention to the striking increase in all tones up to and includ-




RECORD OF CONVERSATIONAL LOSSES

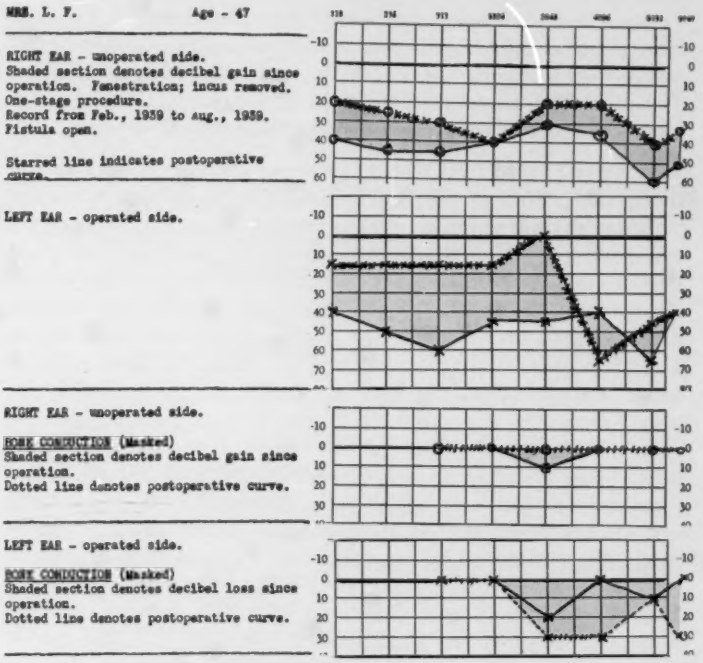
| Date | Right | Left |
|---|---|---|
| Sept., 1938 | 47% | 28% |
| Aug., 1939 | 9% | 15% |

Fig. 14.

ing 2,048. I also call your attention to the improvement in the opposite ear.

There has been considerable discussion as to the need for preserving the incus, presuming it to articulate with an anky-

losed stapes. Its utility as a factor in transmitting sound impulses, on the basis of any of the accepted theories by which we are supposed to hear, I seriously question. Certainly, this case seems to indicate that its preservation is not a necessary factor to gain a great betterment in hearing acuity. That it




RECORD OF CONVERSATIONAL LOSSES

| Date | Right | Left |
|---|---|---|
| Feb., 1939 | 51% | 40% |
| Aug., 1939 | 24% | 8% |

Fig. 15.

may act, as Sourdille suggests, as a fulcrum over which sound impulses impinging upon the drum may be transmitted to the fistula along the tympanomeatal flap, I seriously doubt. The excellent results obtained in the case charted as Figs. 13 and 14, and in this case (see Figs. 15 and 16) raise the question

of the necessity for always retaining the incus and for the absolute need of a flap for the achievement of hearing betterment by fistulization.

Note also in this case that in the highest tones (see Fig. 16) no improvement has occurred, but, nevertheless, the improved hearing for the conversational range is so good that the percentage loss for these tones has improved from a 40 per cent loss to one of only 9 per cent. Nevertheless, as I have pointed out in Fig. 10, there has occurred a loss in the bone conduction (by masking) since operation. This, too, awaits explanation since this case is thus far a success from all standpoints.

In *Case 8*, shown in Fig. 17, I bring another point into the discussion. This is the hearing chart of a patient who, for reasons having nothing whatsoever to do with the topic we are discussing, required an operation which embraced surgical destruction of the membranous horizontal semicircular canal. He made an uneventful recovery, did not develop any postoperative sepsis or labyrinthitis. His hearing acuity both before and after operation are here charted. Note that three months after the operation, his hearing acuity in the operated ear was practically the same as it was before I destroyed the membranous canal. Can we not deduce from this and from other cases recorded in literature that surgical trauma to the membranous labyrinth which is *not followed by infection* need not always mean that hearing will be totally lost? Incidentally, from this finding we may also surmise that a prompt decapsulation of the membranous labyrinth, where an acute hydrops threatens the function of Corti's organ, may be indicated to avert the hearing loss threatened by the developing hydrops. On this point, too, further observations are necessary.

## CONCLUSIONS.

From my material presented, some tentative deductions may be drawn. I agree with Campbell and with Canfield that the tissue destined to cover the fenestration is less important than the *manner of making the fistula.* Contrary to what has been said of the heat caused by the polishing burr, and the warning to avoid this heat, I believe that the heat which the burr produces is one of the most important elements in inhibiting bony closure of the fistula. In some of my best results, as far as

maintaining a patent fistula is concerned, I so worked the burr that sufficient heat was created to produce a thermal laby-

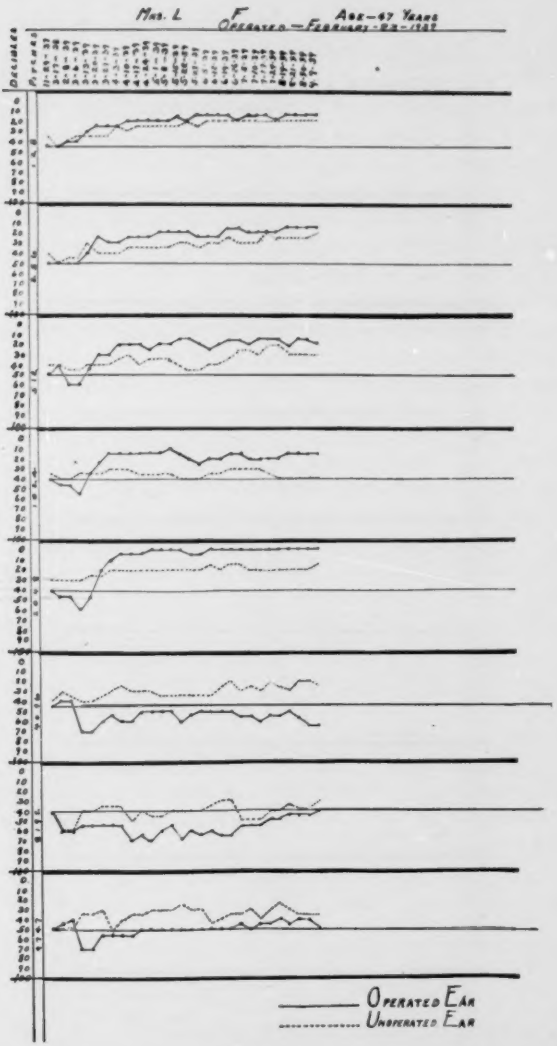



Fig. 16.

rinthine reaction, evidenced by induced nystagmus, long before the telltale dark line had brought the endosteal layer of the

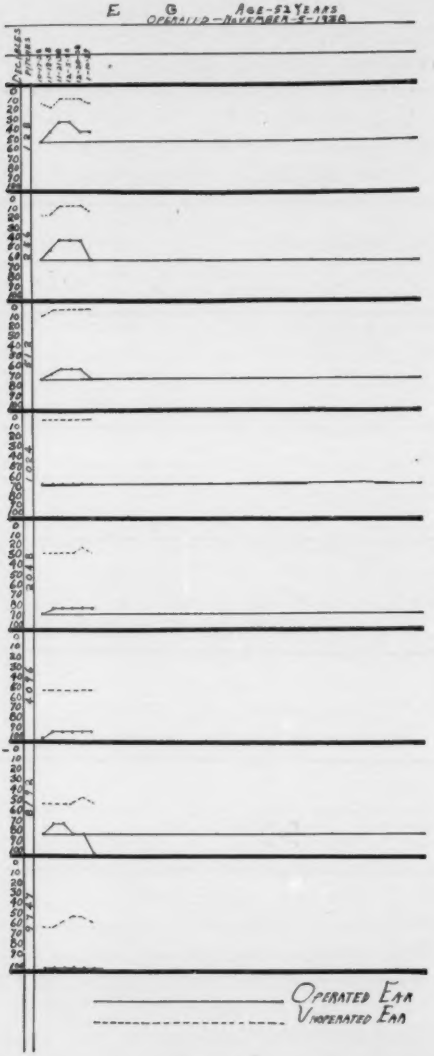



Fig. 17.

canal into view. Where, in the earlier cases, I carefully tried not to produce heat, my results were not as good as when I disregarded this warning and actually strove to produce a degree of local heat with the burr.

The size of the fenestration plays a part, too, in the end-results. In a few cases I made a long exposure of the membranous labyrinth. My results here were not good. My best results have been obtained with a fenestration of from 1 to 3 mm. in length. Definitely, further study is required to determine the extent of the fistula needed in relation to the size of the round window, and to the degree of stapes fixation which is present in a given case. Furthermore, in some instances, even with a persistently open fistula, the hearing gradually drops, and the initial gain is lost. Here I have found that a systemic infection of one or another sort was the inciting cause. Is there not some suggestion here that the causal incident which precipitated the deafness will continue and remain the factor responsible for the defeat of a successful fenestration?

I have presented Fig. 17 to demonstrate that deliberate destruction of the membranous canal need not of itself destroy or reduce hearing. As a matter of fact, in such an event a primary increase in hearing acuity often occurs — only to drop to its previous level. Finally, the data accumulated in my experience tends to bring into question all our accepted theories of the physiology of hearing. The collection of sound wave impulses by the drum, their transfer through an ossicular chain, their impact upon the labyrinthine fluids and then a selective action by a given portion of the organ of Corti — all of this must be reviewed. Neither does the "telephone" theory of hearing supply the answer. The question of an electrical potential in the cochlea which initiates an action-current through the cochlear branch of the VIIIth nerve also falls short of entirely fitting the circumstances presented in these improved cases. Yet I feel that this theory, now under more intensive scrutiny, may hold the answer to our problem.

Diagnostic criteria in habitual use do not necessarily make for a positive diagnosis. In several instances I have uncovered totally unsuspected pathology. When such is found, it must be attacked. The further results from fistulization, then, depend upon the resourcefulness and ingenuity of the surgeon.

I have called attention to the behavior of the unoperated ear in these cases. It deserves study. In my cases wherein a cholesteatome impeded hearing, the unaffected ear was normal. Where, however, a chronic progressive deafness was present, both ears gradually lost hearing acuity. When, in such cases, a succesful fistulization had been accomplished, not only did the operated ear show improvement, but the unoperated ear also. Is it not, therefore, important to revalue the bilateral innervation of the cochlea and its central distribution? Can we not conceive that the awakening of the cortical hearing centres, stimulated by the reactivation of the one ear, through afferent nerve impulses, will increase sensory perception by the unoperated ear? This finding, to my mind, opens up the entire question of the rôle which the brain as a whole, as well as designated parts of it, plays in the function of hearing.

I feel that before operating on these patients, great care should be exercised to learn which ear was first involved in the deafness. That probably is the ear at fault, and I am strongly impressed that the second ear has lost function largely because there has been a lack of stimulation to the association centres over a long period of time, from the initially involved side. I have records of very early cases where only one ear showed the classical symptoms of otosclerosis, and only after an interval of some years did the second ear follow suit. This I have observed particularly in some very young patients.

In closing these brief hints *on the problems involved,* suffice it to say that the problem of affording relief surgically to the deafened *is still far from solution.* I strenuously urge that the surgical work be continued. We shall learn much from careful observations made by competent surgeons. Above all, I trust that physiologists will re-examine, in the light of the data obtained by this surgery, the fundamentals of hearing.

On the question of what permanent value this surgery holds out for the deafened, no final conclusion can be made as yet. More time must elapse. At present, there seems to be reason to hold to the view that something is developing which will be of value in the therapy for deafness.

## REFERENCES.

1. LEMPERT, JULIUS: Improvement in Hearing in Cases of Otosclerosis: A New One-Stage Surgical Technique. *Arch. Otolaryngol.*, 28:1:42, July, 1938.

2. SOURDILLE, MAURICE: New Technique in the Surgical Treatment of Severe and Progressive Deafness from Otosclerosis. THE LARYNGOSCOPE, 47:12:853, Dec., 1937.

3. HOLMGREN, GUNNAR: Surgery of the Ear, 17:399. Thos. Nelson & Sons, N. Y., 1938.

4. NAGER, F. R.: Demonstration zur Holmgren'schen Otoskleroseoperation an Affen. *Acta Oto-Laryngol.*, *27*:4:350, 1939.

5. CANFIELD, NORTON: Labyrinthine Fistulas: Reports on Experiments on the Vital Response to Various Methods of Producing Defects in Bone. *Arch. Otolaryngol.*, 30:1:50, July, 1939.

6. CAMPBELL, EDWARD H.: Report presented to the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 10, 1939.

7. KOPETZKY, SAMUEL J.: Discussion. THE LARYNGOSCOPE, 47:12:911-912, Dec., 1937.

71 East 80th Street.

# THE EXPERIMENTAL PRODUCTION OF DEAFNESS IN YOUNG ANIMALS BY DIET.*†

DR. EDWARD MELLANBY, London, England.

In a series of publications since 1926 it has been shown that degenerative changes in many nerves of both central and peripheral systems of young animals can be readily produced by dietetic means (Mellanby, 1926, 1930, 1931, 1933, 1934b, 1935, 1937, 1938). The most common lesions consist of demyelination changes followed by complete disappearance of the fibres in many of the afferent peripheral nerves and ascending fibres in the central nervous system. It has been shown that nerve cells are also affected by these dietetic conditions and reasons have been given which suggest that these nerve cell changes may be primary to the demyelination of the nerve fibres.

In the usual experimental period of about four months, the afferent fibres degenerate before the efferent, which may not be much affected, if at all. With a longer dietary period the efferent fibres are affected, but never as badly as the afferent.

Although the most obvious lesions consist of changes in the myelin sheath there are also changes in the axis cylinders in the nerves of both dogs and rabbits fed on diets deficient in carotene and vitamin A. These changes can be seen in fibres which show annular myelin degeneration and even in cases where there is no other obvious defect in the nerve. The axis cylinder changes have been studied in more detail in branches of the sensory nerves supplying the dental tissues by King, Lewinsky and Stewart (1938) in vitamin A deficient rats.

The dietetic conditions favoring these developments are: *1.* deficiency of vitamin A and carotene; and *2.* presence of much cereal. Of these two factors, the absence of vitamin A and carotene is the more important, for in young rats, at least, the same demyelination changes can be produced by synthetic diets in the absence of cereal if they are deficient in vitamin A and carotene (Zimmerman, 1933). Again, the cereal content

*Reprinted from the Journal of Physiology, Vol. 94, No. 3, p. 380, 1938.

†From the Field Laboratory, Sheffield University, and the Farm Laboratory, Mill Hill, National Institute for Medical Research, London, England.

of the diet may be very high and yet, in the presence of sufficient vitamin A and carotene, little or no nerve degeneration will be produced. On the other hand, changes in the type of cereal eaten or the addition of certain cereal products may alter the degree of degeneration. The experimental diets are made up of ordinary foodstuffs, except for the irradiated ergosterol, and are rich in all substances of the vitamin B complexes.

One of the afferent nerves affected by demyelination changes under these dietetic conditions is the VIIIth, both cochlear and vestibular divisions, the former more so than the latter. Degenerate changes in the vestibular nerves are associated with inco-ordinate movements of locomotion of the animal, as might be expected. Young dogs showing this condition are also inattentive and do not respond to the call of people at close range, a characteristic which distinguishes them from normal puppies. This inattention is doubtless due partly to deafness.

The present paper is concerned with these abnormal changes due to vitamin A deficiency, and contains a description of the pathological condition of the labyrinthine capsule, both of its nerve supply and the bone itself. When a rich source of vitamin A is added to the diet while every other condition of diet and environment is kept constant, the animals remain normal in behavior and the labyrinthine capsule escapes pathological change.

All young animals examined up to date, *i.e.*, dogs, rabbits and rats, react to the above dietetic defects, so far as nerve degeneration is concerned, in the same way, differing of course in degree. The new facts, especially concerning bone hyperplasia, described in this paper, refer for the time being to the dog. There is, however, no doubt that bone hyperplasia takes place in the rabbit and probably in the rat, and will be described in a future paper.

## HISTOLOGICAL TECHNIQUE.

Since the degenerative changes of a nutritional nature in the VIIIth nerve were first seen and described, the need of following up and examining in detail the internal ear itself has been constantly in mind. Many of the animals fed over a fairly long period on diets very deficient in vitamin A were

obviously deaf, and unsuccessful attempts were made to grade the degree of deafness in dogs and rabbits by observing their reactions to sound. The earlier histological preparations made for the detailed examination of the finer structure of the cochlea and vestibule were unsatisfactory. Last year I discussed the subject of deafness in animals on deficient diets with Mr. Cleminson, Head of the Ferens Institute for the Study of Otology, and he was good enough to arrange for a demonstration of the technical methods for histological examination of the internal ear in use at that Institute. Mr. Peet kindly demonstrated these methods of fixation by perfusion through the heart and gave full details of the further technique; moreover, serial sections of celloidin embedded material were made from two labyrinths fixed by Mr. Peet. These and other sections were carefully examined by Dr. Hallpike, to whom I am most grateful for assistance and advice. The Ferens technique has in the main been used in the present work, with the exception that, in order to shorten the time needed for obtaining the final result, paraffin has replaced celloidin embedding in the end process. In many cases formolsaline was used as a fixative agent instead of Wittmaack's solution, which contains potassium bichromate. For decalcification, 5 per cent nitric acid was often used instead of 1 per cent. By these devices the time occupied in preparation was reduced from four to five months to between five and seven weeks. The resulting histological preparations are not as good as those obtained by the Ferens Institute method, especially as regards nerve cells and epithelial structure, but the great saving of time counterbalances this drawback. As I understand that Dr. Hallpike will publish a detailed account of the Ferens Institute method for the histological study of the labyrinth capsule, no further reference will be made to the methods used in the present work.

A total of 51 labyrinths from 44 dogs have been examined. Serial sections of 16 of these from 12 animals have been cut, stained and examined; in some cases when both labyrinths of one animal were examined, pathological changes were not identical. The whole cochlea and vestibule of the remaining cases were cut but not mounted serially. In addition, ground undecalcified sections were prepared from some of the ears for purposes of bone examination. Some experiments of a similar nature have been made also on rabbits and rats and serial sections of the labyrinthine capsules cut. These experi-

ments will not be described in the present publication. It is now evident that because of the complicated structure of this organ and the multiple changes that occur in the labyrinth under the experimental conditions that it would have bee easier if serial sections had been made from all the experimental animals, in order to appreciate more fully the actual course of events. This, however, was not possible, but the number of labyrinths serially sectioned is probably sufficient to allow of a general description of the changes produced.

## EXPERIMENTAL METHODS AND RESULTS.

The experiments on young dogs described in the present paper were not made primarily to test the effects of different diets on the ear. The action of certain forms of diet in producing and preventing extensive nerve degeneration had been well established and most of the experimental feeding was at the time centered on an attempt to get information on the part played by cereals and cereal products in influencing these changes. This explains why, so far as the cereal portion of the diet is concerned, there are small differences in the various series of experiments. It is not the intention in the present publication to deal with this point, however, since the effect of changing the cereal portion of the diet is only one of degree and not of kind. For the time being, attention will be mostly confined to the changes in the neurones and bones of the cochlea and labyrinth produced by variations in the vitamin A content of diets rich in cereals. Reference will, however, be made to some animals in whose diet potato wholly replaced the cereal.

The experiments were made under the following conditions:

Litters of puppies varying from seven to 10 weeks old were given diets consisting of separated milk, cereal (oatmeal, white flour, etc.), lean meat, yeast, peanut oil, irradiated ergosterol (vitamin D), orange or lemon juice, and sodium chloride. In this diet there is very little vitamin A or carotene, but it is, as far as is known, otherwise complete.

The time necessary for producing the pathological change varies with the stores of vitamin A in the liver of the puppy at the beginning of the experiment, and also with the severity of the dietetic deficiency. Usually, however, the animal's behavior is abnormal after two to four months of such feeding

and the changes may be great after four months. In the experiments reported in this publication the duration of the dietetic period varied from four to 10 months in different animals. The pathological changes were very intense in the dogs which had received vitamin A deficient diets for the longer periods.

Apart from the specific effects produced by the vitamin A deficiency, the animal's health was usually fairly good, the food was eaten well and the weight steadily increased. Dogs do not readily die of infection in these conditions,, as they might be expected to do when one thinks of the multiple infective lesions developed by young rats fed on vitamin A deficient diets. Nor do dogs develop xerophthalmia as easily as do rats, but when they do it is a more serious matter, owing to the rapidity with which perforation of the cornea often takes place. The relation of xerophthalmia to generation of the trigeminal nerve has been previously described (Mellanby, 1934a).

A typical experiment in which advanced degenerative changes were produced will be given first. Three puppies, age 8 weeks, were given the following dietary mixture daily over a period of 42 weeks: separated milk powder, 12.5 up to 30 g.; oatmeal, 75 up to 270 g.; peanut oil, 7.5 up to 15 cc.; meat deprived of visible fat, 25 up to 60 g.; baker's yeast, 3.75 up to 12 g.; sodium chloride, 1 up to 4 g.; vitamin D as irradiated ergosterol, 1,000 I.U. The former quantity represents the amount of each constituent given at the beginning of the experiment, and the latter the amount given when a maximum diet was being eaten. In addition, dog I had daily 30,000 I.U. of vitamin A. Dog II did not get any additional source of vitamin A throughout the feeding period. Dog III received no added vitamin A for the first 15 weeks, but for the last 27 weeks it had 30,000 I.U. daily.

At the end of the experiment, the vitamin A content, measured as Carr-Price blue values, using a Lovibund tintometer, of the livers of the three days were as follows:

| Dog | Carr-Price Blue Values Per G. Liver | Approximate Equivalent in I.U. Per G. Liver |
|---|---|---|
| I (Vitamin A rich diet) ..... | 93 | 2,800 |
| II (Vitamin A deficient diet) .. | Nil | Nil |
| III (Curative experiment) ..... | 50 | 1,500 |

Dog III will not be further discussed here but will be referred to later in dealing with possible recovery.

Fig. 1 represents a section of the cochlea and its components of dog I (vitamin A rich diet). Fig. 2 is a corresponding photomicrograph of dog II (no vitamin A). (Only a few of

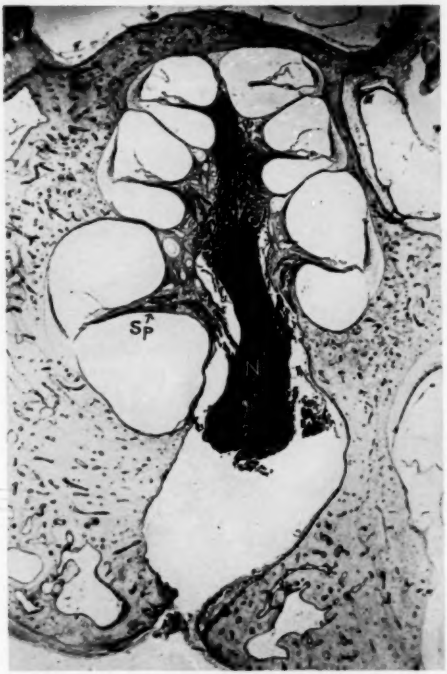



Fig. 1. Lower power photograph (x19) of cochlea of dog I. Basal diet with vitamin A added. On diet 10 months. It will be seen that the internal auditory meatus is clear and the cochlea branch of the VIIIth nerve (N) has an uninterrupted path to the brain. The spiral ganglion cells (SP) are clearly seen (cp. Figs. 2, etc.)

the photomicrographs illustrative of the results obtained were made from dogs I and II. Other illustrations were sometimes chosen, not because they were different, but because they were technically better.)

The most obvious pathological changes found in the labyrinths of young dogs brought up on these vitamin A deficient

diets are: *a.* nerve degeneration, more especially of the cochlear neurones; *b.* new bony growth in the modiolus; *c.* overgrowth of the internal periosteal layer of the capsule, *i.e.*, the bone in proximity to the brain; *d.* serous labyrinthitis; *e.* degenerative changes of the organ of Corti and sensory

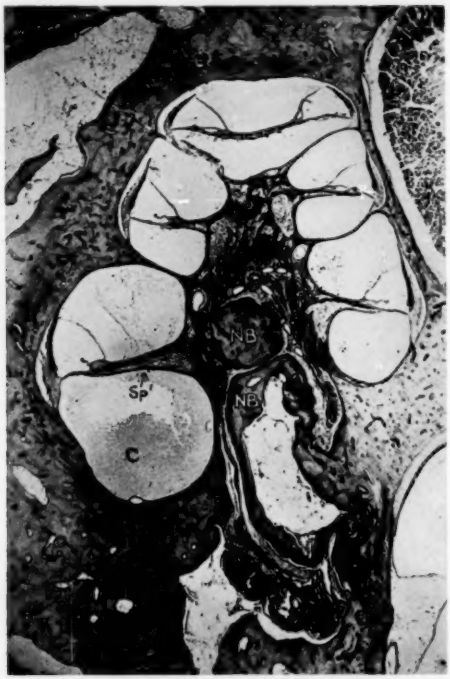



Fig. 2. Low power photograph (x19) of cochlea of dog II. Basal diet as for dog I, but in this case no extra carotene or vitamin A was added. On diet 10 months. Two new bony masses (NB) practically filling the internal auditory meatus can be seen. The cells of the spiral ganglion (SP') have disappeared and are replaced by connective tissue. Albuminoid coagula (C) can be seen in scala tympani, indicating serous labyrinthitis.

epithelium of the semicircular canals. Occasionally a small amount of bony overgrowth was seen in the basal whorl of the scala tympani.

*Changes in the VIIIth (Cochlear and Vestibular Divisions):* The cochlear division (or its remains) of the VIIIth nerve is seen in Figs. 1 and 2. In Fig. 2, representing the cochlea of

dog II (vitamin A deficient diet for 10 months), this nerve has disappeared, whereas in Fig. 1 from dog I (vitamin A rich diet) it is intact. Not only has the cochlear nerve of dog II completely degenerated but the spiral ganglion cells and the nerve fibres to the organ of Corti have gone, and their place

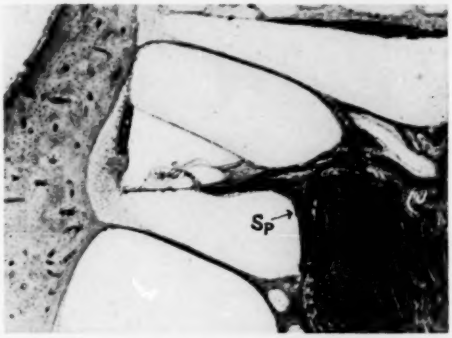



Fig. 3. Middle whorl of cochlea (x50) of a normal dog showing organ of Corti and cells of spiral ganglion (Sp).

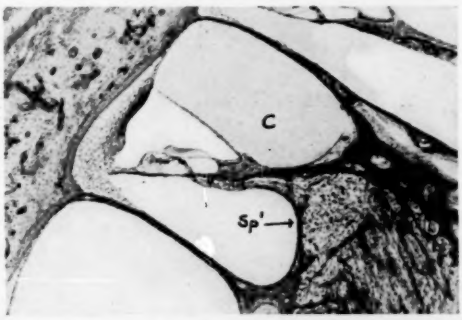



Fig. 4. Middle whorl of cochlea (x50) of a dog on a diet containing much cereal and deficient in vitamin A. On diet five months. Many cells of the spiral ganglion (Sp') have disappeared together with the peripheral branches to the organ of Corti. Indications of serous labyrinthitis can be seen in the scala vestibuli.

is filled with loose connective tissue. The spiral ganglion cells and nerve fibres to the organ of Corti of dog I are normal.

Photomicrographs (x50) of a whorl of a cochlea of each of three dogs are seen in Figs 3-5. Fig. 3 is normal and is in great contrast to both Figs. 4 and 5, which are from dogs fed

on diets deficient in vitamin A. In Fig. 4 the vitamin A deficient animal was killed after five months of the diet. Many cells of the spiral ganglion have disappeared and those remain-

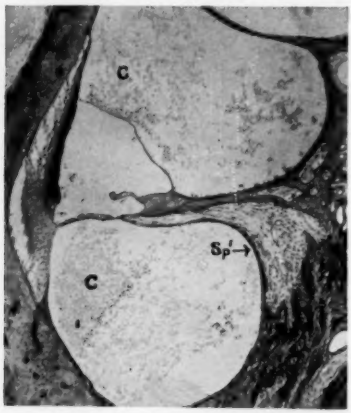



Fig. 5. Basal whorl of cochlea (x50) of dog II. Diet deficient in vitamin A. On diet 10 months. The cells of the spiral ganglion (Sp') have completely disappeared together with the peripheral nerve fibres to the organ of Corti. Albuminoid coagula (C) are obvious in both the scala tympani and scala vestibuli, indicating a condition of serous labyrinthitis. The organ of Corti is very abnormal.

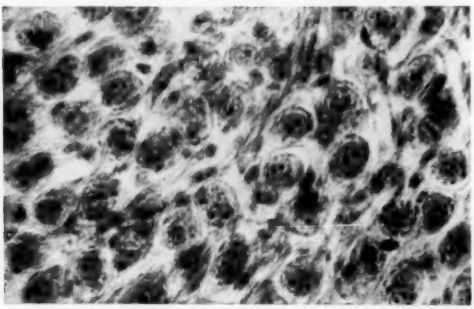


Fig. 6. High power photograph (x500) of cells of the spiral ganglion of a normal dog. Note definite Nissl granules and well defined nuclei.

ing are shrunken and the cytoplasm is free from granules. Most of the peripheral branches to the organ of Corti have also gone. In Fig. 5, taken from dog II, which had been on the vitamin A deficient diet 10 months, the condition is still

worse; there are no cells and no fibres left. Figs. 6 and 7 are high power photomicrographs (x500) showing the detailed condition of the cells of the spiral ganglion in a normal (receiving vitamin A) and a vitamin A deficient animal, respectively. The normal spiral ganglion cells seen in Fig. 6 are very different from those in Fig. 7, which represent part of the spiral ganglion seen in Fig. 4. Comparison between Figs. 3-5 also shows that in the vitamin A deficient animals (see Figs. 4 and 5) the peripheral fibres of the cochlear nerve supplying the organ of Corti have degenerated, whereas in the animal receiving vitamin A (see Fig. 3) they are present.

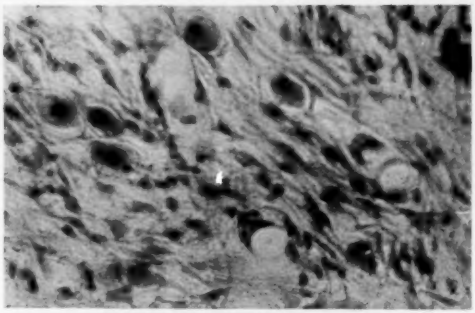


Fig. 7. High power photograph (x500) of cells of the spiral ganglion of a dog whose diet was deficient in vitamin A. Nearly all the ganglion cells have completely degenerated and the few remaining cells are shrunken and the protoplasm is homogeneous. Cytoplasm is devoid of granules.

It will thus be seen that in an advanced case (see Fig. 5) the whole cochlear nerve, including the spiral ganglion cells and their central and peripheral branches, disappears.

In experiments where the nerve degeneration is not as advanced as in the instance just described, the spiral ganglion cells at the base of the helix are usually rather more abnormal than those at the apex; one exception to this rule, where the basal spiral ganglion cells are less affected than the apical cells, was encountered.

Although all the cells of the spiral ganglion of dog II (see Fig. 2) were degenerated, there were still some cells in Scarpa's ganglion. It is true that they were in most cases degenerate but they had not completely disappeared, as had the cells of the corresponding spiral ganglion. Throughout this inves-

tigation it has been found that the cochlear division of the VIIIth nerve is more affected than the vestibular. The ganglion cells of Scarpa's ganglion in dog I (vitamin A rich diet) are normal. These results are in keeping with those previously described (Mellanby, 1935), where it was shown that, comparing the number of degenerating fibres in the cochlear and vestibular divisions of the VIIIth nerve in A-deficient animals, it was always found that the cochlear division suffered more severely than the vestibular division.

*Overgrowth of Bone of the Labyrinthine Capsule:* A glance at Figs. 1 and 2 shows another striking development in the internal auditory meatus of the vitamin A deficient dog (see Fig. 2). It will be seen that two massive pieces of bone fill up

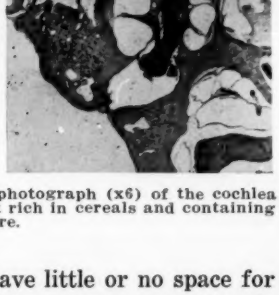


Fig. 8. Low power photograph (x6) of the cochlea and labyrinthine capsule of dog I on a diet rich in cereals and containing vitamin A. Normal or nearly normal structure.

the meatus and leave little or no space for the branch of the VIIIth nerve. The bone appears on the whole to be of normal structure but the lower piece has a large cavity full of fatty tissue. The corresponding meatus of Fig. 1 (vitamin A rich diet) is patent and the VIIIth nerve here has an uninterrupted passage to the brain.

The filling of the internal auditory meatus at the modiolus end seen in Fig. 2 is only found in advanced cases of degeneration produced by the prolonged dietetic deficiency described, but all experimental animals so far examined brought up for a few months on similar diets show some such bony change.

Another position where bony overgrowth is found is in the periosteal bone adjacent to the brain and to the internal auditory meatus. This new bone can be seen by comparing Figs. 8,

9a and 9b, which are low power photographs of the labyrinthine capsule of dog I (see Fig. 8) and dog II (see Figs. 9a

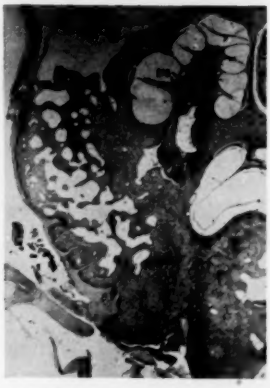


Fig. 9a. Cochlea and labyrinthine capsule (x6) of dog II on a diet rich in cereals and deficient in vitamin A. Not only is the internal auditory meatus nearly filled with new bone (NB) but the periosteal bone of the capsule, especially on the brain aspect,, is greatly increased in thickness, the new bone being cancellous in type. The exit of the VIIIth nerve is not seen in this section.

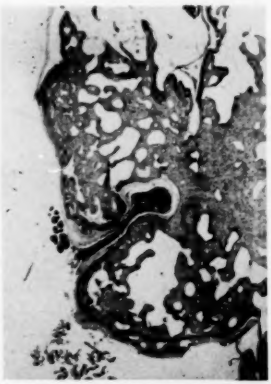


Fig. 9b. The section represented here is about 2.5 mm. from that shown in Fig. 9a. The exit of the VIIIth nerve can now be seen. The two photographs (see Figs. 9a and 9b) when compared with the normal (see Fig. 8) illustrate the tortuous course, the compression and stretching of the auditory nerve associated with the bony overgrowth in dogs brought up on a vitamin A deficient diet.

and 9b). In Fig. 8 (diet rich in vitamin A) the meatus and the cochlear division of the VIIIth nerve can be traced from

the helix of the cochlea to the edge of the capsule. In Fig. 9a the meatus is tortuous and the exit of the nerve from the brain cannot be seen, so that several sections are needed to trace its full course. This displacement is illustrated in Figs. 9a and 9b; Fig. 9b, where the exit of the VIIIth nerve or its remains is clearly seen, represents a section about 2.5 mm. from that shown in Fig. 9a.

The depth of bone between the basal whorl of the cochlear helix and the brain is much greater in Fig. 9a (from A-deficient animal) than in the control (see Fig. 8). The cancellous nature of the new bone is also clearly seen in Figs. 9a and 9b. This increase in periosteal bone is more clearly demonstrated in Figs. 10 and 11 representing sections cut in a plane at right angles to those of Figs. 8, 9a and 9b. It will be seen that whereas in the normal dog (see Fig. 10) the bone on each side of the internal auditory meatus is thin, in Fig. 11 it is greatly thickened, due to the laying down of new periosteal bone, which appears to be of the ordinary cancellous type. There is no excess of osteoid tissue, nor is there any evidence of the changes being inflammatory in origin. The spaces in the bone are abundant in outer periosteal layer and are usually filled with fat.

The effect on the nerves of these two bony overgrowths, the one in the modiolus filling the upper part of the internal auditory meatus, and the second resulting in the thickening of the periosteal layer, must be harmful. The first squeezes the nerve; the second greatly lengthens the distance from the respective ganglia of the cochlear and vestibular divisions of the VIIIth nerve and thus stretches the nerve.

Compression not only of the cochlear but also of the vestibular division of the VIIIth nerve by new bone formed in and near the modiolus can be well seen in Fig. 12a, which represents the cochlea of a dog after five months on a vitamin A deficient diet. In this figure a smaller mass of new bone ($NB^1$) is present in the modiolus above the spiral ganglion at the base of the helix, and a second larger mass ($NB^2$) between the division of the cochlear and vestibular branches of the main nerve. The spiral ganglion cells have largely degenerated (see Fig. 7 and compare with the normal in Fig. 6), but in the case of the cells of Scarpa's ganglion, although many of these are squeezed into an abnormal shape, they have not

degenerated (see Fig. 14 and compare with the normal in Fig. 13).

It has been mentioned above and elsewhere (Mellanby, 1935) that the cochlear division of the VIIIth nerve is more liable to be completely destroyed by these nutritional condi-

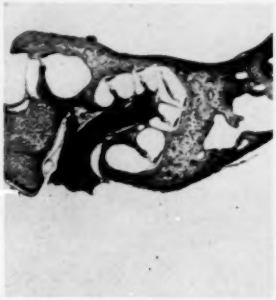


Fig. 10. Labyrinthine capsule (x6) of a normal dog, but in a plane at right angles to Figs. 8, 9a and 9b.

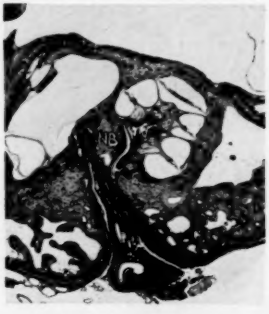



Fig. 11. Labyrinthine capsule (x6) of a dog on a diet rich in cereals and deficient in vitamin A. There is again a mass of new bony growth in the modiolus (NB) and great thickening of the periosteal layer of the labyrinth capsule, causing compression and elongation of the nerve. Cf. Fig. 10, normal section cut in same plane.

tions than the vestibular division, and yet both branches are compressed and stretched by these bony overgrowths. This possible difference in susceptibility to mechanical influences will be considered later.

*Serous Labyrinthitis:* Besides the nerve and bone changes described above, the condition known as serous labyrinthitis

was also produced by these diets deficient in vitamin A. The albuminoid coagula in the perilymph spaces which are characteristic of this condition can be seen in Figs. 4 and 5. In the normal animal (see Fig. 3) no such coagula can be seen. A

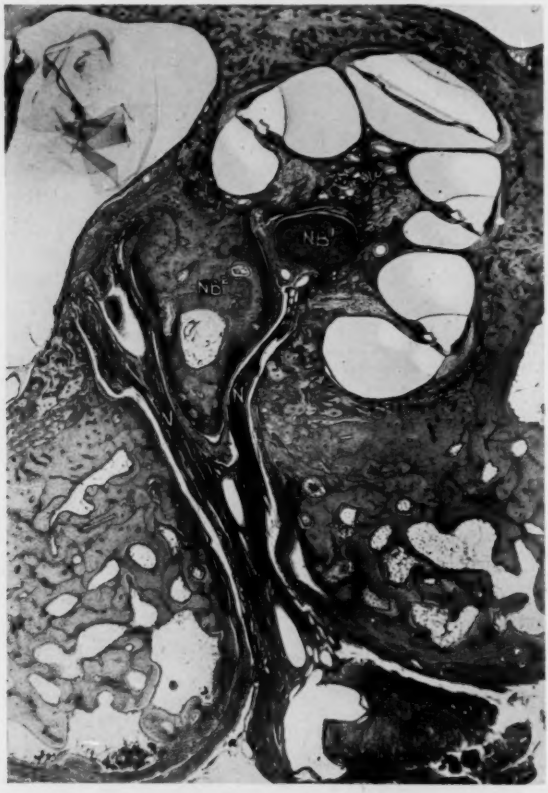



Fig. 12a. Bony overgrowth ($NB^1$ and $NB^2$) causing compression of both the cochlear and vestibular branches (N) of the VIIIth nerve in a dog on a cereal diet deficient in vitamin A (x19). Only a few degenerate cells remain in the spiral ganglion but more are found in Scarpa's ganglion (see Figs. 13 and 14).

similar pathological condition is well known in man. There is no evidence in the present experiments that the process is infective in origin, nor indeed that any of the changes described is the result of inflammation.

What the relation is between the serous labyrinthitis in the vitamin A deficient animals and the nerve and bony changes

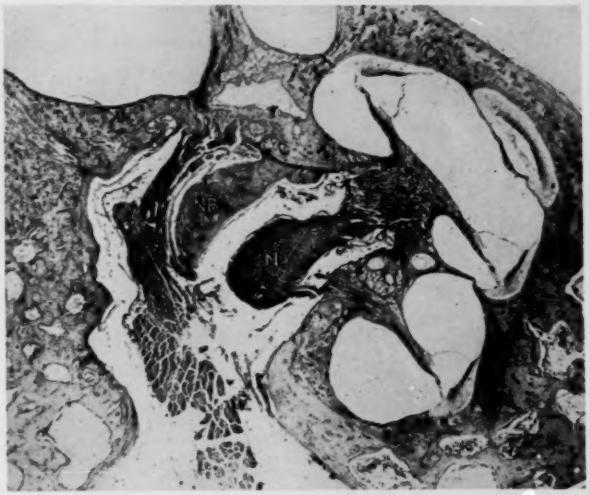



Fig. 12b. Cochlea (x19) of a dog maintained on a diet deficient in vitamin A for five months. The diet was similar to that of the previous case (see Fig. 12a), except that potato replaced the cereal. The cells of the spiral ganglion are only slightly degenerate. There is some new bone in the modiolus (NB) and slight thickening of the periosteal bone. The changes seen in this animal on a potato diet are much less advanced than those of the animal on a cereal diet for the same period (see Fig. 12a).

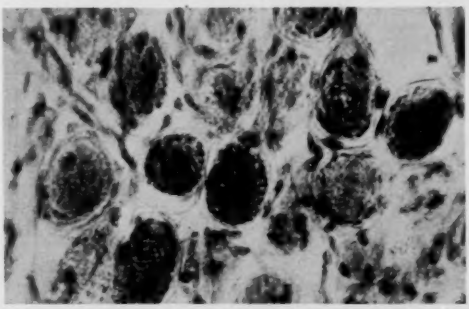


Fig. 13. Cells of Scarpa's ganglion (x500) of a normal dog.

cannot yet be stated. It is possible that the abnormality of the perilymph is due to the spread of some toxic influence

from the subarachnoid space of the posterior fossa up the cochlear aqueduct, as this is a common pathway for the transit of toxins in serous labyrinthitis in man (Hallpike, 1937). This point is being further investigated.

Serous labyrinthitis in man is also associated with a series of degenerative changes in the organ of Corti and the sensory epithelium of the vestibule. Similar changes take place in the animals under discussion.

*Degenerative Changes in the Organ of Corti:* In serous labyrinthitis in man, one of the earliest changes in the organ of Corti is the loss of differential staining power between cytoplasm and nuclei in the cells of the rods of Corti and in the

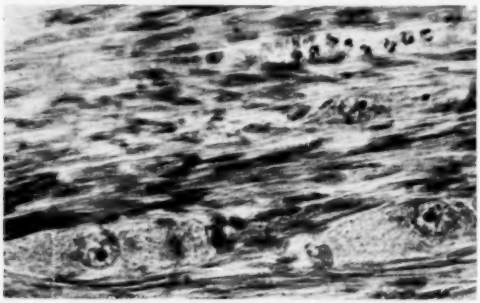
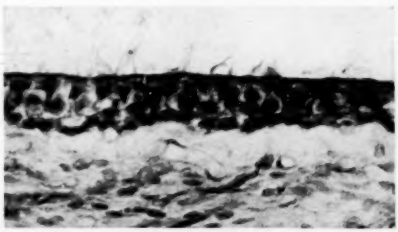


Fig. 14. Cells of Scarpa's ganglion (x500) of a dog on a diet rich in cereals and deficient in vitamin A (see Fig. 12a). Although the cells are comparatively normal in structure, they are markedly elongated, apparently due to the pressure of the new bone (NB) shown in Fig. 12a. These cells seem very resistant to mechanical injury as compared with those of the spiral ganglion.

outer and inner hair cells. This change cannot be seen in the histological preparations so far made in the present work, probably because the technique used is not suitable. In severer cases of experimentally produced serous labyrinthitis, the degeneration of these cells becomes more marked, the hair cells lose their hairy projections and ultimately these auditory cells, as well as Deiter's and other cells of the organ of Corti, disappear, leaving a small amount of undifferentiated tissue lying on the basement membrane (see Fig. 5). In spite of the degeneration of these cells, there still remains the small tunnel of Corti. In Fig. 3, representing the organ of Corti of a dog receiving vitamin A, the structure is normal.

*Degenerative Changes in the Epithelial Cells of the Ampullae:* Up to the present time a comparative study of the vestibular apparatus has not been made but changes in the epithelium of the semicircular canals in those animals fed for long periods on A-deficient diets are also obvious. Comparing Figs. 16a and 16b (vitamin A deficient) with Figs. 15a and 15b (receiving vitamin A), it will be seen that there is a

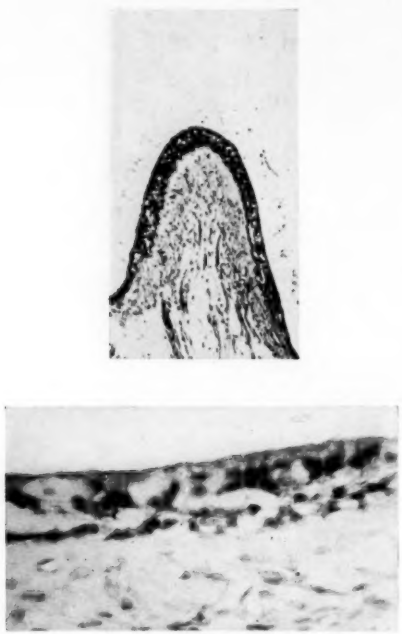


Figs. 15a and 15b. Low power (x50) and high power (x300) photographs of ampulla of dog I on a diet rich in cereals and vitamin A. The epithelium is thin, there is good differentiation between the nuclei and cytoplasm of the cells and the hair-like projections are clearly seen.

general thickening of the epithelium in the former. Associated with this thickening there is a loss of differentiation of the individual cells, the disappearance of the nuclei and a shedding of hairy projections.

*Relation of Serous Labyrinthitis to End-Organ Degeneration:* When the degenerative changes in the organ of Corti and in the sensory epithelium of the vestibule above described

were first noted, it was thought that these might be due to degeneration of the cochlear and vestibular nerves, respectively. Dr. Hallpike informed me, however, that this was not the case, since there was good evidence that cutting the VIIIth nerve, although it produced degeneration of the whole cochlear neurone, did not produce degenerative changes in the organ of Corti. He also informed me that serous labyrinthitis



Figs. 16a and 16b. Low power (x50) and high power (x300) photographs of ampulla of dog II on a cereal diet deficient in vitamin A. The epithelium is thickened, the differentiation between nuclei and cytoplasm is poor, and the hairy projections have in many places disappeared.

in man ultimately affected the organ of Corti and the sensory epithelium of the ampullae. It seems likely, therefore, that this is the sequence of events in the present experiments.

Serous structural changes in the organ of Corti are a late event compared with the nerve degeneration and bone hyperplasia and appear always to be accompanied by serous labyrinthitis. On the other hand, in shorter experiments there

may be serous labyrinthitis without large structural defects in the organ of Corti. There is also a suggestion from one result in the present work that serous labyrinthitis can be cured by adding vitamin A to the diet even after the bone overgrowth and nerve degeneration are advanced. It is likely, for instance, by analogy with the other experimental results, that dog III (curative experiment) had developed serous labyrinthitis as well as nerve and bone changes at the end of the 15 weeks on a vitamin A deficient diet. This dog received abundant vitamin A for the next 27 weeks and at the end of this period the cochlear neurone had almost completely degenerated. There was, however, little or no serous labyrinthitis nor was there any abnormality of the organ of Corti or sensory epithelium of the ampullae of the semicircular canals. It would appear possible from this experiment that the addition of vitamin A, after a period of vitamin A deficiency, cleared up the serous labyrinthitis and so prevented the degeneration of the organ of Corti but did not prevent the process of destruction of the cochlear neurone. This possible explanation would be in keeping with the view that degeneration of the sensory epithelium of the labyrinth in these experiments is due to serous labyrinthitis and not to degeneration of the auditory nerve. Further experiments to settle this point are, however, necessary.

Serous labyrinthitis is probably not so early a pathological development as bone overgrowth or nerve degeneration, although it is true that wherever bone hyperplasia and nerve degeneration are advanced (apart from the curative experiment of dog III) serous labyrinthitis exists. In early and slighter cases of bone and nerve abnormality there may be no serous labyrinthitis. This is best seen in experiments in which the cereal element of a vitamin A deficient diet is replaced by potato. The amount of bone hyperplasia in such cases is comparatively small though definite, and the cells of the spiral ganglion are slightly degenerated, but there is little or no serous labyrinthitis and the cells of the end-organs are apparently normal (see Fig. 12b).

*Early Changes in Labyrinth Due to Dietetic Abnormality:* As some of the results, especially the bony overgrowth, were quite unexpected, the appearance in earlier stages of the abnormality has not so far been studied, although histological

material is now being prepared from experiments of shorter duration.

These early changes can, however, be well seen in a series of experiments in which the cereal portion of the diet was replaced by potato. The diets otherwise were as previously described. It has been already pointed out (Mellanby, 1934b) that replacement of cereal by potato in these vitamin A deficient diets greatly reduces the degree of nerve degeneration. What may be the real explanation of this effect is not known—it is possible that cereals have a toxic action and hasten the pathological changes in the absence of vitamin A and that their replacement by potato reduces this action, or it may be that the small amount of carotene in potato is sufficient to prevent the development of the more pronounced changes. It may be added, however, that the dogs eating the potato diets had no stores of vitamin A in the liver at death. This point will not be considered further here.

After a period of five months on the potato diet deficient in vitamin, the animals were very active and comparatively normal in appearance and behavior, and contrasted greatly with those on a similar diet but containing cereal. Histological examination of the labyrinthine capsule at the end of this period usually shows a small amount of bone overgrowth in the modiolus and increased periosteal bone; this can be clearly seen in Fig. 12b. How relatively small the bone overgrowth is can be seen if comparison is made with Fig. 12a, which represents the labyrinth of a dog maintained on a cereal diet deficient in vitamin A for the same period (*i.e.*, five months) as the potato-fed dog of Fig. 12b.

The amount of nerve degeneration in this latter dog is slight but definite. There is actually only a small amount of degeneration in the cochlear neurone (central fibres) but the spiral ganglion cells appear abnormal to some extent.

Little or no serous labyrinthitis is present in these cases and no degenerative changes of the organ of Corti or the sensory epithelium of the vestibule can be seen.

*Overgrowth of Bone at the Base of the Skull Obvious to the Naked Eye:* After microscopic examination had revealed the large bony overgrowth produced by these vitamin A deficient diets it was soon obvious, even to the naked eye, that the

base of the skulls of these animals was greatly deformed by this overgrowth. In addition to the auditory nerve, it was evident at once that other nerves, including the optic, trigeminal and facial, were pinched by the bony overgrowth and that this mechanical pressure might well explain the degenerative changes of these nerves previously described. This matter will be considered in more detail in a subsequent publication.

*Rabbits and Rats:* That there are also changes in rabbits and rats fed on vitamin A deficient diets there can be no doubt, but details of the abnormalities have not been studied sufficiently to warrant a description here. In the vitamin A deficient animals so far examined, there are abnormalities, including nerve fibre and cell degeneration, bony overgrowth and serous labyrinthitis.

## DISCUSSION OF RESULTS.

Similar experiments previously published (Mellanby, 1934b, 1935) had shown that the central branches of the cochlear and vestibular divisions of the VIIIth nerve in the labyrinthine capsule were degenerated to some degree, the cochlear fibres more than the vestibular. The present work confirms this earlier work and allows a much better judgment to be made, both of the degree of degeneration and the condition of the various parts of the cochlear neurone. In some cases the whole cochlear division had degenerated and the animals must have been completely deaf. In such cases the vestibular division was also severely affected but not to the same degree, and even when the cells of the spiral ganglion had completely disappeared it was usual to find some cells of Scarpa's ganglion remaining.

The surprising result of the present investigation was the new bone formation in the labyrinthine capsule which accompanied the nerve degeneration. So far as the writer knows, there is no previous evidence that vitamin A deficiency results in bone overgrowth. This new bone, so far as the labyrinth is concerned, affects the periosteal bone near the brain and is not found in the deeper layers away from the brain. It is of cancellous nature with marrow cavities usually full of fat. It is normal in appearance and is not of inflammatory origin. Indeed, the capsules of these affected dogs are apparently free from infection in the ordinary sense. This point is mentioned

because it will be remembered that rats brought up on vitamin A deficient diets often have infected middle ears (Green and Mellanby, 1928).

It has been pointed out that the new bone laid down appears constantly in two places, although in each it is of periosteal origin. The first of these positions is in and near the modiolus itself. This bone may appear more compact and usually only one or two cavities, sometimes large, are obvious in any one section. This bone can be seen in the photomicrographs to press on and elongate the nerves as they leave the cochlea and vestibule, respectively. The second position of bone overgrowth is round the internal auditory meatus and adjacent to the brain. It has the effect of placing the cochlea deeper into the capsule and of increasing the distance from the spiral and Scarpa's ganglia to the brain. This must stretch the nerve. In addition, however, this bony overgrowth sometimes twists the internal auditory meatus, so that it may be impossible to get a complete section of the nerve in one histological preparation, as can usually be done in normal animals. This twisting of the internal auditory meatus must also stretch the nerve.

New bone may also occasionally be seen in a third place, namely, in the scala tympani at the basal whorl of the helix. The inconstancy of the small amount of bone in this third position indicates that it is probably independent of the other two masses of periosteal bone and may rather be related to serous labyrinthitis.

The general result of this investigation, therefore, is that a deficiency of vitamin A in the diet has resulted in the development of newly formed bone and the degeneration of the VIIIth nerve, especially the cochlear neurones. As a rule, the greater the bone formation the greater the degeneration of the nerves of the labyrinth, and the conclusion that the nerve degeneration is due to mechanical interference produced by bone overgrowth is difficult to avoid. This deduction is supported by what is already known of the reaction of each division of the auditory nerve to injury of their central branches. If the cochlear nerve reacted as do most sensory nerves to injury of its central branch, it would not be expected that the cells of its spiral ganglion and their peripheral branches should be so easily destroyed by mechanical influences bearing on its

central branch in the internal auditory meatus. It is known, however, that the cochlear nerve is unlike most other sensory nerves in this respect and is even unlike the vestibular division. For instance, Wittmaack (1911) showed that destruction of the central branch of the VIIIth nerve in the internal auditory meatus with preservation of the blood supply caused degeneration of the cells of the spiral ganglion with their peripheral branches to the organ of Corti. Wittmaack also found that the peripheral vestibular branch and Scarpa's ganglion did not degenerate after this operative procedure, although the central branches of the vestibular nerve were destroyed. These results have been confirmed by Kaida (1931), by Hallpike and Rawdon-Smith (1934) and Hallpike (1938). Other confirmation comes from the observations of Crowe (1929), corroborated by de Kleyn and Gray (1932), that pressure upon the VIIIth nerve by a tumor in the internal auditory meatus leads to degeneration of the peripheral cochlear neurone.

These facts show that the degenerative reactions of the cochlear nerve represent a notable exception to Waller's law. It is clear also that the condition of the cochlear neurone in the present work is in keeping with its degeneration being due to pressure and stretching of the central fibres by the newly formed bony masses.

In contrast to the great susceptibility of the cochlear nerve to compression and other injuries of its central branch, the resistance of the vestibular nerve to compression and elongation, which has been demonstrated above (see Figs. 4 and 5), is noteworthy. The experiments of other workers have also demonstrated this fact, for cutting the central branch of the vestibular neurone, as in the case of most other afferent nerves, produces only temporary derangement of Scarpa's ganglion and no degeneration of the peripheral branch (Hallpike, 1937). Thus the better preserved condition of the vestibular nerve in comparison with that of the cochlear nerve again supports the view that the nerve destructive changes are mechanical in origin and are due to bone overgrowth.

The point under discussion, namely, whether bone overgrowth is directly responsible for the degeneration of the VIIIth nerve, is of fundamental importance and obviously cannot rest at the present point. The nerve degeneration pro-

duced by these vitamin A deficient diets is widespread and includes not only the VIIIth nerve but also the optic and trigeminal nerves, the posterior roots of the spinal cord and many ascending fibres in the cord, including the endogenous nerve fibres of the anterior and posterior spinocerebellar tracts. If, therefore, bone overgrowth is responsible for the death of the auditory nerve, it would be expected that the degenerative change in all the other nerves, peripheral and central, would have a similar cause. It is, therefore, desirable that the evidence that the auditory nerve has been killed in these experimental animals because of the overgrowth of bone should be as clear as possible, and that the possibility that the bone overgrowth is secondary to degeneration of the nerve or that both bone overgrowth and nerve degeneration are independent of each other but dependent upon some third factor should not be excluded.

On the whole, the evidence of the present experiment that the overgrowth of bone of the labyrinthine capsule is responsible for the death of the nerves is as clear as can probably be expected. It is hoped that further experiments in which slight degrees of change are produced will help to settle the point at issue. Even so, however, the present results demanded that the investigation should be extended to see how the other nerves of the brain and spinal cord, which are also known to degenerate under these nutritional conditions, are affected by bone overgrowth in their immediate neighborhood.

This work is now being done and it is already clear that there is much bone hyperplasia in the neighborhood of the optic and trigeminal nerves. Indeed, the skull in immediate contact with the base of the brain, including the bone surrounding the various foramina, is undoubtedly deformed in dogs brought up on the vitamin A deficient diets. Early examination, therefore, supports the view that the degeneration of other nerves of the brain may well be due to the bone overgrowth as in the case of the auditory nerves. It would be expected, therefore, that a similar mechanism holds in the case of the nerves of the spinal cord, and further examination is being made on this point. It is difficult at first sight to see how mechanical factors due to bone overgrowth can destroy endogenous fibres of the central nervous system, such as those of the anterior and posterior spinocerebellar tracts, but this

point may be clarified by further investigation. In the meantime, so far as the present work goes, it seems certain that the degeneration of the auditory nerve brought about in young dogs by diets deficient in vitamin A and made up of ordinary foodstuffs is due primarily to excessive bone formation of the periosteal bone of the labyrinthine capsule and the mechanical interference of this new bone with the auditory nerve.

There remains for consideration the relation between serous labyrinthitis present in the vitamin A deficient dogs and the bone and nerve changes above described. But little can be said of this at the present stage of the work. It is probable that serous labyrinthitis is not the earliest change, since in some dogs showing only slight abnormalities, as for instance in the animals receiving potato in their diet instead of cereal, although there is definite bone hyperplasia and early nerve degeneration, there is little or no serous labyrinthitis. In such cases albuminoid coagula are not obvious in the perilymph and the organ of Corti is normal in appearance. On the other hand, where the bone overgrowth and nerve degeneration are advanced, there is always serous labyrinthitis. It looks as if the bone and nerve changes precede the development of labyrinthitis in the earlier period of pathological change.

Another difference between the nerve degeneration and serous labyrinthitis is suggested by the appearance of the labyrinth of dog III (curative experiment). In this animal the cochlear neurones had completely disappeared, but there was no labyrinthitis. By comparison with other experimental animals it seems likely that this animal also had serous labyrinthitis at the end of 15 weeks on a vitamin A deficient diet. If this were the case, then the addition of vitamin A for the following 27 weeks must have cleared up the condition. It has not, however, prevented the nerve degeneration from going on to completion. Reasons have been given above for thinking that the degenerative changes in the organ of Corti and the sensory epithelium of the ampullae of the semicircular canals are associated with the development of serous labyrinthitis and are not related to the nerve degeneration. It was pointed out, for instance, that the cochlear neurone may have completely disappeared, and yet in the absence of serous labyrinthitis the organ of Corti may appear normal or nearly normal.

Finally, it may be asked, how do these vitamin A deficient diets produce all these changes in the labyrinthine capsule?

This question cannot be answered at present, but it is already clear that the answer must include not only consideration of the auditory mechanism but of the many other nerves in the body. Although it is probable that overgrowth of new bone holds the secret to the sequence of pathological changes, in the cranial nerves at least, this part of the subject has not been sufficiently investigated to allow a full description to be given, except in the case of the auditory nerve. On the other hand, a fairly complete survey of nerve degeneration produced by these diets has been worked out in the case of the dog and rabbit (Mellanby, 1934b, 1935), and it is now necessary to see whether all such degenerative changes in the spinal cord as well as the brain can be related directly or indirectly to overgrowth and pressure of bone. Whether this is so or not, it is already evident that overgrowth of bone in young animals due to vitamin A deficiency does not affect all bones. In the long bones the absence of vitamin A probably affects the texture of the cancellous tissue but it does not appear to alter the shape of these bones.

In view of the historical development of knowledge of bone growth, especially in regard to rickets and the discovery of the calcifying vitamin D, it is of peculiar interest that vitamin A, with which vitamin D is so often associated, should also be concerned with bone formation. The parts played by these two vitamins are, however, essentially different. In the absence of vitamin D there is overgrowth of osteoid tissue in all growing bone. In the absence of vitamin A there is overgrowth of apparently normally formed bone in certain places. In view of the growth-promoting function usually assigned to vitamin A, it is of interest to note that its presence in young dogs *prevents* overgrowth of the labyrinthine bone. It would be fruitless at this stage to conjecture the nature of the mechanism whereby vitamin A works in limiting the growth of bones at the base of the skull and other places to their normal shape, so that in its absence these bones overgrow and destroy the nerves. It is certain, however, that the explanation of this mechanism must depend on interesting physiological processes of whose nature nothing is known at the present time.

## SUMMARY.

*1.* The histological examination of the labyrinth capsules of young dogs fed for some months on diets of natural foodstuffs

but deficient in vitamin A and rich in cereals revealed the following changes:

*a.* Degeneration of different degrees up to complete disappearance of the cochlear nerve, the cells of the spiral ganglion and their central and peripheral branches.

*b.* Degeneration, but to a lesser degree, of the vestibular division of the VIIIth nerve.

*c.* Overgrowth of bone in the modiolus and of the periosteal layer of the capsule near the brain.

*2.* This overgrowth of bone is apparently responsible for the degenerative changes in the nerves by reason of the pressing and stretching of these tissues.

*3.* Serous labyrinthitis also develops in the cochlea of the dogs on a vitamin A deficient diet. This condition seems to produce degeneration of the sensory epithelium of the labyrinth, including that of the organ of Corti and of the ampullae of the semicircular canals, in course of time.

*4.* Substitution of potato for the cereal element of these vitamin A deficient diets greatly reduces the abnormal changes in the labyrinth above described.

*5.* Examination of the base of the skull of these vitamin A deficient dogs reveals other bone overgrowth and deformity, which is probably responsible for the degenerative changes of other cranial nerves, such as the optic and trigeminal nerves, previously described.

The expenses of this investigation were paid by the Medical Research Council, to whom my thanks are due.

## REFERENCES.

CROWE, S. J.: *Arch. Surg.*, Chicago, 18:982, 1929.

DE KLEYN, A., and GRAY, A. A.: *Jour. Laryngol.*, 47:589, 1932.

GREEN, H. N., and MELLANBY, E.: *Brit. Med. Jour.*, 2:691, 1928.

HALLPIKE, C. S.: *Jour. Laryngol.*, 52:661, 1937.

HALLPIKE, C. S.: *Jour. Path. Bact.*, 46:216, 1938.

HALLPIKE, C. S., and RAWDON-SMITH, A. F.: *Jour. Physiol.*, 83:243, 1934.

KAIDA, Y.: *Jap. Jour. Med. Sci.*, 12:237, 1931.

KING, J. D.; LEWINSKY, W., and STEWART, D.: *Jour. Physiol.*, 93:206, 1938.

MELLANBY, E.: *Jour. Physiol.*, 61:24 p., 1926.

MELLANBY, E.: *Brit. Med. Jour.*, 1:677, 1930.

MELLANBY, E.: *Brain*, 54:247, 1931.

MELLANBY, E.: *Edinb. Med. Jour.*, 40:197, 1933.

MELLANBY, E.: *Jour. Path. Bact.*, 38:391, 1934a.

MELLANBY, E.: *Nutrition and Dis.* 1934b. Edinburgh and London: Oliver and Boyd.

MELLANBY, E.: *Brain*, 58:141,1935.

MELLANBY, E.: *Chem. Ind.*, 56:1054, 1937.

MELLANBY, E.: *Jour. Physiol.*, 93:42 p., 1938.

WITTMAACK, K.: *Verh. dtsch. otol. Ges.*, 20:295, 1911.

ZIMMERMAN, H. N.: *Jour. Exp. Med.*, 57:215, 1933.

38 Old Queen Street.

# THE USE OF "SECONAL" IN MINOR PROCEDURES IN OTOLARYNGOLOGY.*

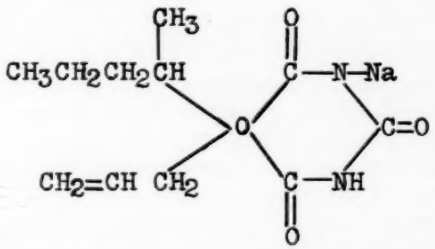
DR. E. H. LYMAN, St. Louis.

## INTRODUCTION.

In otolaryngology there are many procedures in which some degree of anesthesia or analgesia is needed. There are also many instances in which preoperative apprehension should be allayed. This discussion is limited to a consideration of the use of "Seconal" in these conditions.

## STRUCTURE AND FORMULA.

Seconal is sodium propyl methyl carbinyl allyl barbiturate. Its structural formula is:



This compound contains an unsaturated aliphatic group and a secondary amyl group. The chemical structure and preparation have been worked out in detail by Shonle and his associates.[1, 2, 3]

## PROPERTIES.

The properties of the drug as a short-acting hypnotic have been studied by Swanson[4, 5] on various laboratory animals. It was found that, in general, seconal had a smaller minimal anesthetic dose and a smaller minimal lethal dose than other comparable drugs, such as sodium amytal and pentobarbital

*From the Department of Otolaryngology, Washington University School of Medicine.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 21, 1939.

sodium, a briefer period of hypnosis, a more rapid recovery time and as wide a safety margin.

Further pharmacological data, particularly as applied to human beings, have been determined by Kempf,[6] who has studied the clinical use of seconal extensively. He found that the duration of anesthesia with seconal was longer than with Pentobarbital sodium, and comparable to that of sodium amytal, but that the time necessary for recovery from seconal was approximately half that required for sodium amytal, and slightly less than that required for pentobarbital sodium. Kempf has used seconal in cases of delirium, mastoiditis with meningitis, for analgesia for spinal puncture, light anesthesia for opening abscesses, and paracentesis. He has also used it in obstetrical cases, in diabetics for rectal surgery and in many purely medical cases. Kempf used dosages of approximately 0.5 to 0.75 gr. per 10 pounds of body weight. He states that excitement is more pronounced in adults than in children, respirations are slowed and blood pressure lowered in similar degree to pentobarbital sodium. The white blood count and blood sugar remain unchanged.

Kempf presents the following criteria as apparent indications: *1.* In small doses, as a fleetingly active sedative to induce sleep. *2.* As an anesthetic for minor surgery. *3.* For hypnosis in labor and as a preanesthetic for delivery. *4.* As a preanesthetic for all general anesthesias when it is desired that the hypnotic effect shall last only during induction of general anesthesia. *5.* Its use as a basal anesthetic, to outlast general anesthesia, would necessitate repeated doses.

Frech and Volpitto[7] recently reported favorably on the use of seconal plus scopolamine for obstetrical anesthesia.

## OBSERVATIONS.

In the past year seconal has been used in approximately 100 cases for various purposes on the otolaryngological services at Barnes and St. Louis Children's Hospitals. In adults it has been used chiefly as preoperative medication orally for local tonsillectomy, the usual dose being 3 to 4.5 gr. In children we have used it for various procedures, as follows: *1.* Preoperatively in tonsillectomy under general anesthesia to decrease apprehension and facilitate induction of ether.

*2*. For paracentesis. *3*. For irrigation of maxillary sinuses. *4*. For lumbar puncture and spinal manometer tests, where restlessness and incomplete relaxation of the patient could easily alter the results of the examination. *5*. As a prelminary medication for submucous elevation of the nasal septum under local anesthesia for the control of epistaxis in rheumatic fever. *6*. For retrograde dilatation of the esophagus. *7*. for adenoidectomy in one case of neurofibromatosis with destruction of the cervical vertebrae and consequent danger of severance of the spinal cord, in which a general anesthetic could not be given. *8*. For a simple mastoidectomy under local anesthesia in one case of postscarlet mastoiditis in which a general anesthetic was contraindicated.

## DISCUSSION.

The usual method of administration in children is by rectum. The seconal in starch base is put into suspension in one or two ounces of water and given as a retention enema, 15 to 30 minutes before the proposed procedure. It was found that usually greatest analgesia and amnesia were obtained in 20 minutes. The seconal can, if desired, be put into as little as 5 cc. of water for rectal administration. In children the dosage varied from 0.75 gr. to 3 gr., estimated according to the age and weight of the patient. In general, it was found that seconal produced adequate analgesia for the procedures in which it was used. The effect was prompt. There was not a prolonged narcosis after the procedures were finished, but usually a brief natural sleep, from which the child would awaken without nausea or other ill effects. Seconal did not prolong the reaction time after a general anesthetic. In some instances an insufficient dose was given, and a few children did not get satisfactory analgesia even with larger doses up to 4.5 gr. There have been no serious ill effects; however, as in any case in which a sedative is used in conjunction with a general anesthetic, when seconal is used preliminary to ether, the patient must be watched carefully until he has reacted to insure proper position of the lower jaw and tongue to allow unobstructed respiration.

Probably the most interesting case in which seconal was used is the child on whom a mastoidectomy was performed.

This was a boy, age 5½ years, who had contracted scarlet fever three weeks prior to admission to the hospital. Ten days before admission, both ears became painful and began to discharge. In the next few days, postauricular swelling developed on both sides, more on the right, and the child soon had marked loss of hearing. On admission, the child was obviously extremely ill, with a temperature of 39° C., markedly dehydrated, apathetic and completely deaf. Both eardrums were red and bulging. There was a perforation in each drum, nipple-like on the right side, each with thick, pulsating discharge, and edema of the external canal walls. The nose was filled with pus, the lips were dry and crusted, the tonsils were very large and moderately inflamed. Sulfanilamide was started on the day of admission. On the same day, bilateral paracentesis was done, the child being given 2 gr. of seconal per rectum 30 minutes before the procedure. Thick pus was obtained. The following day, after restoration of body fluids, a simple mastoidectomy was done on the right side, using 3 gr. of seconal rectally 30 minutes before the operation and 0.5 per cent novocain in the skin along the line of incision as local anesthesia. The entire mastoid was necrotic and there was also a Bezold abscess. Improvement was rapid following the operation. Eleven days later, diagnostic antral irrigation was done, no pus being found. This was done with 2 gr. of seconal given rectally. Two days following this, a simple mastoidectomy was done on the left side under gas-ether anesthesia, the child's general condition being sufficiently improved to permit a general anesthetic. After the second mastoidectomy the patient's temperature ran a septic course for 12 days, although his general condition remained good. At the end of this time, a right-sided cervical adenitis was explored and no pus was found; however, there was much necrotic tissue and a drain was inserted. This was followed by cessation of fever and progressive recovery. Six weeks after admission, the tonsils and adenoids were removed under ether. The child was discharged two months after admission with the left ear completely dry, minimal discharge in the right ear and the hearing much improved. In this one case seconal was used to great advantage three times.

Appreciation is expressed to the members of the hospital staffs who kindly co-operated and assisted in this study.

## CONCLUSIONS.

Our experience in the use of seconal as an analgesic and amnesic in otolaryngological procedures at Barnes and St. Louis Children's Hospitals indicates that this drug is a valuable and safe one to use. In many instances it eliminates the necessity for a general anesthetic; and when given preliminary to a general anesthetic, such as ether, allays apprehension and facilitates the induction.

## REFERENCES.

1. SHONLE, H. A., and WALDO, J. H.: A Study of the Effect of Unsaturated Aliphatic Groups in Barbituric Acids. *Jour. Amer. Chem. Soc.*, 55:4649, 1933.

2. SHONLE, H. A.: Barbituric Acids Containing a Secondary Amyl Group. *Jour. Amer. Chem. Soc.*, 56:2490, 1934.

3. SHONLE, H. A., and KLEIDERER, E. C.: Mixed Melting Point Curves of Some Dialkylbarbituric Acids. *Jour. Amer. Chem. Soc.*, 56:2489, 1934.

4. SWANSON, E. E.: Sodium Propyl-Methyl-Carbinyl Allyl Barbiturate; a Short Acting Hypnotic. *Proc. Soc. Exper. Biol. and Med.*, 32:1563, 1935.

5. SWANSON, E. E., and FRY, W. E.: A Comparative Study of Two Short-Acting Barbituric Acid Derivatives. *Jour. Amer. Pharmac. Assn.*, 26:1248, Dec., 1937.

6. KEMPF, G. F.: Personal Communication.

7. FRECH, H. C., JR., and VOLPITTO, P. P.: Amnesia in Obstetrical Practice. A Study of Sodium Pentobarbital (Nembutal)-Scopolamine and Sodium Propyl-Methyl-Carbinyl-Allyl Barbiturate (Seconal)-Scopolamine. *Jour. Med. Assn. Ga.*, April, 1939.

Barnes Hospital.

## THE REMARKABLE SOJOURN OF AN ASPIRATED FOREIGN BODY.

DR. C. E. PURCELL, Paducah, Ky.

The history of the progress and development of bronchoscopy since 1897 is spectacular and no chapter in medical history is more fascinating. The possibilities of bronchoscopy and the dramatic results could not have been anticipated at that time. Since that date, the achievements of bronchoscopy are also a matter of record. A survey of the history during this period shows no reference to a reported case of an aspirated foreign body going through the upper air passages and passing out through the chest wall between the ribs.

No wonder, therefore, that I was surprised on learning of this unusual behavior of an aspirated body, and still more surprised that there does not seem to be a history of such a case during the last 42 years.

In a personal conversation with Dr. F. H. Russell, of Wickliffe, Ky., he related an unusual experience with a pneumonia case. I was immediately very much interested in what he told me, and at my request he furnished the history of a foreign body which was extruded through the chest wall after a very severe attack of pneumonia, which I am here detailing; he also furnished the foreign body itself for my collection.

On May 12, 1938, M. B., female, age 4 years, of Wickliffe, Ky., swallowed a piece of grass. Dr. Russell saw the patient at once and, as there were no symptoms of difficulty of breathing and the child had no dyspnea, he concluded that probably the grass had passed on into the stomach. The child's parents gave him the history that the child had some wild grass in her mouth on her way home from Wickliffe. After a violent choking spell and some coughing, she seemed to be in no distress, and Dr. Russell thought the patient would have no further trouble. On May 17, or five days after the incident, Dr. Russell was called and found the child had pneumonia. There was a very high temperature, ranging up to 105°, with the usual cough and expectoration of large quantities of pus. On the

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 28, 1939.

twenty-eighth day there developed on the right side what appeared to be an abscess of the skin. Dr. Russell informed the parents that an abscess had occurred and that he would probably have to open it the next day; however, before he could do this, the abscess ruptured spontaneously, and there

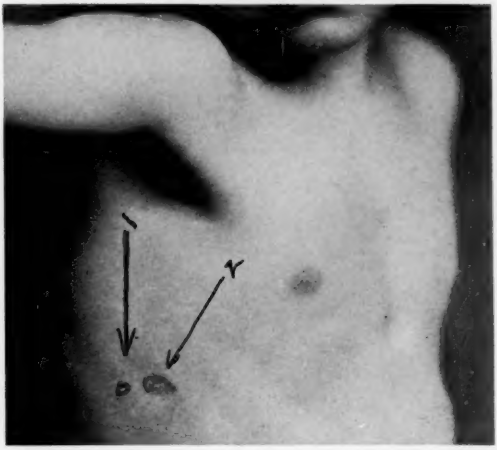


Fig. 1. The accompanying picture of the child shows where the foreign body came out. Dart No. 1 indicates the scar which resulted from the passing out of the foreign body. Dart No. 2 indicates a scar resulting from a former superficial abscess.


Fig. 2. This photograph shows the length of the foreign body as shown by the picture of the rule.

appeared in the opening the end of the piece of grass. The father, with a small pair of tweezers, took hold of this and removed a whole head of grain, the stem end coming out first. This occurred 29 days after the incident, and the head of grain was removed from the abscess on the right chest wall,

between the sixth and seventh ribs. Immdiately following this, the child became much improved and recovered fully very promptly. At my request, Dr. Russell sent his little patient to my office on Nov. 20, 1938. The child appeared very

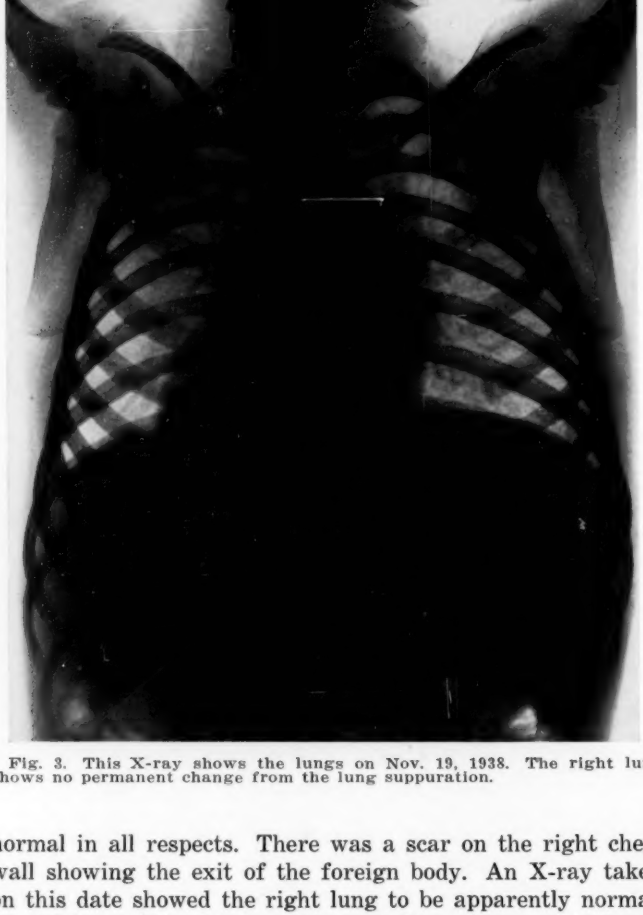


Fig. 3. This X-ray shows the lungs on Nov. 19, 1938. The right lung shows no permanent change from the lung suppuration.

normal in all respects. There was a scar on the right chest wall showing the exit of the foreign body. An X-ray taken on this date showed the right lung to be apparently normal. The picture of the foreign body will give you an exact idea of its size. In the picture of the child there are two scars; the

larger one, according to the patient, occurred some time ago as a result of a boil.

## COMMENT.

This report naturally brings up the question as to why many foreign bodies are not extruded through the chest wall. The answer is that most foreign bodies do not possess the physical properties of anchorage. It is very evident that a foreign body, such as a watermelon seed, orange seed, or a nail, and particles of peanuts and other kernels could never go through the chest wall, because they would have nothing to anchor them at the time of coughing. In this particular case of the head of grain, every cough propelled the foreign body forward; the beards anchored it fast in its new location, just as a jack would do in raising an automobile wheel. The head of grain could not have gone down other than stem first, in the same manner that fish swallow other fish head-first. It is easily understood that a fish could not swallow another fish tail-first, because the fins would necessarily interfere with the progress. It is very evident that the child had pneumonia, which developed into a lung abscess. At each cough, the foreign body was pushed further into the abscess; new abscesses formed, and the same process pushed the grain on through the bronchi, pleura and chest wall.

Guthrie Building.

# TWO CONGENITAL NASAL DEFORMITIES: BIFID NOSE AND BULLDOG NOSE.

DR. MORTIMER M. KOPP, Brooklyn.

Ontogenetic variations in the human face are not frequent. A review of the literature reveals relatively few cases. One of the cases reported in this article, "the bulldog nose," was first described by Trendelenberg. Later, Bumba and Luckst, in 1927, reported this type of deformity at length. Dobryzaniecki also reported two cases and summarized the cases on record.

## THE EMBRYOLOGICAL BACKGROUND.

Stated briefly, the congenital factors involved in bifid nose are:

*1.* The characteristic epithelium of the olfactory placodes is distinguishable during the third week of embryonal life — 4 to 5 mm. embryos.

*2.* In the following week, the nasal areas are depressed by a marked thickening of the circumjacent mesoderm, which, in effect, raises the ectoderm into an inverted "U." This gives rise to what is known as the lateral and median nasal process (12 mm.). The median processus fuse with the maxillary processus of the first arch to form part of the upper jaw. The lateral ones fuse with the maxillary process to form the cheeks, lateral part of lips and alae of the nose. The median nasal processus fuse to become the median part of the upper lip. The mesial remainder of the original frontal nasal process in the interim is compressed, to become septum and dorsum of the nose, respectively. The epithelial plates which separate the nasal fossae from the primitive mouth cavity become thin membranous structures caudally and, rupturing, produce two internal nasal openings, the primitive choanae. Cranially, the epithelial plate is destroyed by ingrowing mesoderm of the maxillary process forming the primitive palate. This latter forms the lip and the maxillary palate. The nasal fossae now open externally through the external nares

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 5, 1939.

and internally into the roof of the oral cavity through the primitive choanae.

*3.* At the termination of the second month of embryonal life, the external framework of the nose is easily recognizable.

*4.* An early failure of normal fusion of preosseous and precartilaginous tissues in the development of the embryo results in one of the many types of deformities.

A bifid nose, or cleft nose, is a congenital deformity in which the distinguishing feature is a separation of the halves of the nose by a furrow. It may appear as a single deformity or in association with other types.

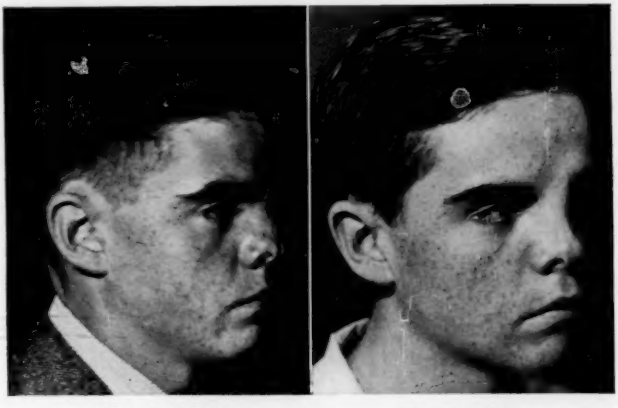


Fig. 1. Before. Fig. 1. After.

D. B. (see Fig. 1), operated at the Israel Zion Hospital, presents an extremely short nose with a distinct "pig snout."

On examination there were found: *a.* A congenital deficiency of the nasal bone (right side). *b.* A congenital deficiency of the frontal bone (right side). *c.* A malposition of the frontal process of the superior maxilla (right). *d.* A compensatory growth of the left frontal, left nasal and frontal processes of the superior maxilla, with a lateral displacement of these bones. *e.* The right facial aspect presented a markedly curved depression, extending downward almost to the nasal tip, which was evident on palpation of the glabella area.

*f*. The curvature in the central portion of the nasal dorsum continued down to the very tip. *g*. The lower, lateral cartilages at the tip were unusually prominent, forming a distinct winged deformity. The "wings" extended deeply on both sides of the cartilaginous septum, giving, at a casual glance, the appearance of a double nose. *h*. The nostrils were flattened and widened.

### THE RHINOPLASTIC CORRECTION.

A previous submucous resection corrected the deviated septum. An accessory cartilage, flat against the septum, was observed. It was readily separated and removed.

The skin over the dorsum of the nose was undermined through intranasal incisions over both lateral cartilages. The columella and the lateral cartilages were next separated from their septal attachments. The lateral cartilages were found to be loosely attached to the sides of the septum.

On intranasal manipulation, the right nasal bone and frontal process of the maxilla were found to be extremely thin, and crackling to the touch. The left side of the nose, contrariwise, was found to be of greater density and, consequently, fractured with difficulty.

Allowance was made in the removal of the nasal hump for the depression on the right side. The saw was then carried through the base of the frontal process of the left maxilla; the fracture and medial displacement were completed manually.

The alar domes were realigned through incisions at the vestibular rim by loosening the cartilages from their attachments to the overlying skin, denuding the skin surfaces of the lower lateral cartilages and then overlapping and sewing them together in order to obliterate dead space.

All circumjacent redundant tissue was exsected to permit of close approximation. The septal mucosae proper on both sides were stripped and the septum thinned by the removal of one of its two layers of cartilage. The loose ends of the septal mucosae were then reattached to the columella.

The nose was pulled down to permit lengthening, and an increase of three-eighths of an inch was thus obtained.

The lateral cartilages were realigned over the septum.

The dressing consisted of adhesive strips and a nasal splint.

A contrasting view of both accompanying pictures shows an obvious marked improvement with the complete removal of the double nose effect.

*Case 2:* L. M., Unity Hospital (see Fig. 2): This patient presented several congenital defects. The facies was typically "bulldog." The frontal processus of the maxillae and the nasal

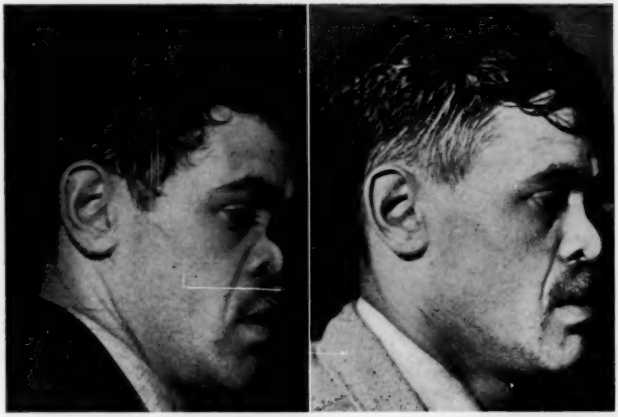


Fig. 2. Before. Fig. 2. After.

bones were rudimentary and formed a flat bridge across the pyriform aperture. The lateral cartilages were absent. The upper margins of the ala dome were attached to the lower free margins of the aperture. The columella was extremely short, causing the nostrils to buckle and arch unnaturally. There was an atresia of the right nasal chamber. The right vestibule ended blindly about three-eighths of an inch behind the external nares. The opening into the left nasal chamber, postvestibular, was pin hole in size. The vestibules communicated with each other because of the failure of union between the columella and the septum.

### THE RHINOPLASTIC CORRECTION.

Under local infiltrative anesthesia, incisions were made intranasally, circumbscribing the pyriform facial aperture, and the nasal tip in its entirety was detached from the underlying bone. The skin covering the blind opening in the right nasal chamber was elevated from the underlying bony attachment. A small opening was curetted in the bone and the introduction of a probe revealed a cavity which appeared to be a preternaturally large maxillary antrum.

The small opening to the left nasal chamber, which readily admitted a probe, was enlarged sufficiently to permit the introduction of a nasal speculum. The nasal chamber appeared to be of normal configuration.

Incisions were made on the upper lip contiguous with and parallel to the columella to permit its lengthening. An increase of three-eighths of an inch of the columella was obtained in this manner.

The skin over the entire dorsum of the nose was undermined.

A previously shaped large piece of ivory was inserted into the nose, under the skin, to restore the profile line and to lengthen the surgically-detached nasal tip. This was kept *in situ* by iodoform packing for four weeks and then replaced by a smaller one.

No attempt was made to close the postcolumellar hiatus between the nostrils.

### BIBLIOGRAPHY.

BOURGUET: La correction esthetique des diverses deformations nosale Les. Norbert Maloine, Editeur, Paris, 1927.

BUMBA and LUCKSCH: Ein Fall von Doggennase. *Virchow's Arch.* 264:2, April, 1927.

CHRYSSIKOS, JEAN B.: Etude de quelques corrections plastiques de l'auvent nasal. *These de Doctorat,* Libraire Louis Arnette, Paris, 1927.

DOBRZANIECKI: Chirurgie plastique et esthetique du visage. *Paris Chirurg.,* No. 5, 1928, and Vol. 48, 1936.

DOBRZANIECKI: Plastic Surgery of the Face. *Rev. de Chirurgie plastique,* 1931.

DOBRZANIECKI: Restoration of the Subseptal Portion of the Nose. *Ann. Surg.,* Dec., 1929.

DOBRZANIECKI: Nowse abiegi wytworcze w zakresie ruchomej ezesci nosa. *Polski Chirurgiczny*, 8:2, 1929.

DOBRZANIECKI: Plastyki twarzy. *Polska Stomatolgja*, Nos. 4, 5, 6, 1931.

JOSEPH: Nasenplastik und sonstige Gesichtsplastik. Curt Kabitsch, Edit., p. 476, Leipzig, 1931.

79 Argyle Road.

---

## THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The Sectional meetings of the Society are scheduled as follows:

Eastern Section, Pittsburgh, Jan. 5.

Southern Section, Columbia, S. C., Jan. 8-9.

Middle Section, Kansas City, Mo., Jan. 19.

Western Section, Los Angeles, Jan. 26-27.

Programs will be mailed to the Fellowship in December. Indications point to a series of excellent meetings.

Subject to confirmation by the Council the annual meeting in 1940 will be held in New York on June 6, 7 and 8; the annual meeting in 1941 in Los Angeles, between May 15 and June 15.

Dr. Hurd has the program for the 1940 annual meeting practically completed. It will include papers on many interesting and timely subjects. For further information, address Dr. C. Stewart Nash, Secretary.

# THE RELATION OF CHEST CONDITIONS TO SINUS DISEASE—THE OTOLARYNGOLOGIST'S POINT OF VIEW.*

DR. AUSTIN T. SMITH, Philadelphia.

The importance of the co-existence of infection in the paranasal sinuses and the lower respiratory tract has been demonstrated both clinically and experimentally with sufficient frequency that it is now accepted as a common clinical fact. Among the first to mention it was Sir St. Clair Thomson[1] in 1914, when he wrote: "Nasal and sinus suppuration often explains the inveteracy of bronchorrhea." An early clinical contribution was that of the French army officers, Rist,[3] Sergent[2] and Saylor, in 1916. Working with supposedly tuberculous soldiers, they reported that in one-third of the cases the diagnosis of pulmonary tuberculosis was incorrect. They found that these patients had a chronic bronchitis associated with chronic infection of the sinuses. They emphasized, as we do now, that the history as to sinus disease was often negative; and that in all cases of chronic or persistent cough a careful examination of the sinuses is most important.

The experimental work of Mullin[4] and Ryder in 1921, and later that of Fenton and Larsell[5] in 1936 has demonstrated clearly the means by which infection of the sinuses can be transmitted to the bronchial and pulmonary tissues. Four possible routes have been demonstrated.

*1.* The trachea by inhalation or direct continuity. This is the easiest and most direct, and seems to me, in the light of clinical experience, quite adequate without the other three.

*2.* The combined path of the lymph nodes, tracheal lymph duct and blood vessels through the right side of the heart and the pulmonary bed.

*3.* The purely hematogenous path.

*4.* Lymph spaces and channels in the visceral cervical space, the dorsal wall of the esophagus, the prevertebral fascia and

*Read at the Meeting of the College of Physicians of Philadelphia, Section on Otolaryngology, Jan. 18, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1939.

related structures, which communicate with the anterior part of the mediastinum.

No direct lymphatic connection between the plexuses and nodes of the head and neck, and the lungs and pleura has been demonstrated.

Confirmation of the early clinical observations of Rist and Sergent has been offered repeatedly by many observers in all parts of the world. In the opinion of most of them the relation is more than coincidental.

Some observers express the opinion that sinus infection may occur secondary to bronchopulmonary infection. Kern and Schenck[6] made observations on patients with post-tonsillectomy abscess of the lung, in whom the sinuses were originally clear on both clinical and Roentgen examination, but in whom within a period of weeks a purulent pansinusitis developed.

Churchill[7] expresses the opinion that the constant spraying of the nasal chambers by coughed-up purulent sputum accounts for the frequent co-existing sinus infection in bronchiectasis.

Mullin[8] objected to this concept of infection spreading from the bronchi to the sinuses, because of the great number of patients, especially children, with cough and moist rales at the bases of the lungs, who get well after early diagnosis of the condition and treatment of the sinuses. Wassan and Waltz[9] drew similar conclusions from Roentgen findings for children with sinus infection and pulmonary disease. Hodge,[10] in an experiment upon 20 patients with bronchiectasis, observed at the Montreal General Hospital, found that during coughing the soft palate seemed a fairly efficient barrier to the entrance of iodized poppy-seed oil and silver protein into the nasopharynx, after these substances had been placed in the trachea. Only a few showed any stain in the nasopharynx, and none was found in the nose.

Whether the sinusitis precedes, follows or develops simultaneously with the bronchial infection may be difficult to settle in certain instances; however, from the practical standpoint of treatment it is of little consequence, for experience teaches that little can be accomplished in the clearing up of

a lower respiratory tract infection with an active focus present in the paranasal sinuses above.

The prevailing concept is that sinusitis and bronchitis develop simultaneously during an acute infection, such as measles or influenza. The bronchitis tends to get well unless fostered and fed by a sinus infection. The repeated aspiration of infective material into the bronchi from the upper respiratory tract is adequate explanation for the persistence of the concomitant lesions. This is logical and is now recognized pretty generally by the majority of writers on this subject.

The frequency of sinus infection associated with infection of the bronchi varies with different reports. Clerf[11] found evidence of sinus disease in 82.4 per cent of 200 cases. Kistner[12] found sinusitis in all but six of 196 cases of chronic nontuberculous bronchitis. McLaurin[13] expressed the opinion that the association of bilateral bronchiectasis and paranasal sinus disease is almost constant. In an analysis of a group of 217 patients with bronchiectasis, excluding patients with congenital bronchiectasis or with bronchiectasis due to foreign body, Walsh and Myer[14] found 66.8 per cent had associated sinusitis. Sinusitis in this group was diagnosed only when gross pus was demonstrable in one or more of the paranasal sinuses. Numerous reports of similar studies found in the literature, all show the same high percentage of co-existing sinus infection and nontuberculous bronchopulmonary disease.

Comparing numerous clinical studies, we find that they agree in certain details. The maxillary and ethmoid sinuses are most frequently involved. Generally speaking, the more extensive bronchial disease is accompanied by extensive sinus disease; often a pansinusitis. Chronic pansinusitis, or gross bilateral infection of the antrum, was found to exist in 53.8 per cent of the cases in the study of Walsh and Myer; however, no direct relationship between the degree of sinusitis and the degree of bronchial disease could be established.

No definite evidence has been presented that there is any connection between the side of the chest affected and the location of the sinusitis. It is notable that the incidence of sinusitis is much lower in cases of bronchiectasis involving only one lobe than it is where more than one lobe is affected.

Another interesting comment from the examination of the various clinical studies of these cases is that the involvement of the right lung shows no predominance of disease over that of the left lung. This is in direct contrast with cases of bronchopulmonary disease due to foreign body, where the right lung is more frequently involved. There appears to be a slight predominance of males over females in the incidence of the associated infections.

No significant facts have been brought out in the bacteriologic studies. The streptococcus appears to predominate in the cases of bronchiectasis and associated sinus infection. In a study of a small group of these patients upon whom I operated radically for extensive disease of the maxillary sinuses and ethmoids, it was interesting to obtain a different organism from a culture of the membrane removed at operation, and the culture of the pus found in the sinus by irrigation prior to operation. Streptococcus hemolyticus, often combined with streptococcus viridans, was obtained from a culture of the membrane in six consecutive cases; whereas, the culture from the purulent exudate showed staphylococcus albus or aureus or some other organism of an indifferent variety. This indicates why an autogenous vaccine may often be useless when it is made from cultures obtained from the sinus washings or purulent exudate found in the nose.

Another interesting feature noted by numerous authors presenting group studies of this condition is its early onset. In many instances chronic sinusitis and advanced bronchiectasis occur in very young children. It apparently is a disease combination of early life.

Goodale,[15] in a recent clinical analysis of 75 cases of bronchiectasis from the viewpoint of sinus infection, found that most of them acquired the disease before the age of 20 years, and nearly a third before the age of 10 years. Most of his patients acquired bronchiectasis following an upper respiratory infection. Twenty-one patients had pneumonia at the onset, which was not preceded, as far as they know, by an upper respiratory infection. An interesting point of differentiation between the upper respiratory and the pneumonia group was that the cases which gave an upper respiratory etiology occurred more frequently between the ages of 10 and 20 years. In contrast to this, the pneumonia group had its

highest incidence in the first decade of life. Also, upper respiratory disease appeared to continue as the causative factor later in life than any other disease. One must bear in mind, in a clinical analysis of a group of cases of this type, that the patients who had pneumonia at the onset of the bronchiectasis may also have had an unrecognized sinus infection. Campbell,[16] in a study of 130 patients with pneumonia, in a consecutive, unselected group, varying in age from 3 weeks to 90 years, showed indication of the presence of sinusitis in 100 per cent.

Clerf[17] states that the greatest field of usefulness in the conservative treatment of bronchiectasis lies in treating the young, and also that the most important factor is the prompt recognition and appropriate treatment of the frequently associated nasal sinus infection.

The significance of the fact that the condition is frequent in early life is that we should be all the more alert for its detection, for in children the greatest good can be expected from conservative measures. The treatment of established bronchiectasis is surgical, and if much is to be hoped for from medical treatment it must be commenced in early childhood. The possibility of a co-existing sinusitis should be suspected in every child who has frequent attacks of bronchitis and pneumonia. The facts that have frequently been brought out indicate that potential bronchiectasis exists where there is co-existing sinus infection and frequent bronchitis.

Clerf,[17] Mullin[18] and others lay great stress on a group of cases, principally children, in which cough is the outstanding symptom. There is a history of recurring colds, developing more frequently during the winter, but the patient is never completely free from cough. The cough is often unproductive. A history of the usual childhood infections, such as measles or whooping cough, is elicited. Symptoms referable to the nose are indifferent or absent. Frequent chest colds are common, and often there is a history of several attacks of bronchial pneumonia which have come and gone; but the outstanding feature has been the persistent cough. It is often so persistent and intractable to ordinary treatment that the patient and the family become used to it, and accept it as a matter of course. Physical examination reveals signs of a chronic bronchitis. Roentgen ray studies show chiefly heavy peribronchial

shadows, principally about the hilum. Diagnostic bronchoscopy reveals a chronic low grade bronchitis, with scant tenacious secretion. There is widening of the carina and of the angles of division of the bronchi. Bronchiectasis is often suspected by the clinician, but cannot be demonstrated by the Roentgenologist or the bronchoscopist. A complete and careful examination of the upper respiratory tract brings to light a chronic infection of the paranasal sinuses, which has been unsuspected. This furnishes the clue to the cause and the essential part of the treatment. This type of case is common, and in the light of our present knowledge of associated chest and sinus infection must be considered as a potential case of bronchiectasis. Brilliant results are secured by prompt recognition and treatment on the part of the rhinologist and internist. The institution of proper treatment, directed to the sinuses and chest, will clear up the chronic tracheobronchitis before bronchiectasis develops.

Although the importance of the relation of chest condition to sinus disease is generally appreciated, and certain facts have been well established, there are still sufficient unknown factors about the relationship to warrant continued investigation. The part that allergy may play in sensitizing the whole tract to infection now appears as a new and interesting angle.

Mullin[18] in his review in September, 1932, expressed the opinion that asthma and bronchiectasis were in no way comparable, and whatever views one may have as to the influence of infected sinuses upon bronchial asthma, they have no place in a discussion of sinus and chest infections; however, at the present time, it is pretty well established that a very important factor in the production of chronic sinusitis is the swelling of the nasal mucosa, incident to nasal allergy; and the reaction of the lower respiratory tract is much the same. Anspach[19] expresses the opinion that allergy may aid in bringing about initial bronchial obstruction by swelling of the mucous membrane and production of a thick, tenacious secretion. Such reaction must develop suitable soil for subsequent infection of the lower respiratory tract. The constant overflow of secretion from the sinusitis is sufficient to keep the bronchial condition active. Hobart Reimann[20] expresses the view that a sensitization of the whole respiratory tract to

infection may exist in some persons. In these persons, the sinuses, bronchi and lungs may be simultaneously involved, or perhaps the infection may spread by continuity. This may occur in childhood and account for the early onset.

Another interesting point of view, that at least illustrates that the problem is anything but a simple one, is that brought out by Sippe.[21] He suggested that hypoglycemia and ketosis may play a part in chronic disease of the antrum and bronchi in both children and adults. He reported nine cases of bronchiectasis; three early cases in which complete relief of symptoms occurred, and six advanced cases in which there was considerable improvement following the administration of dextrose. Sippe suggested that many of the persons who have chronic sinusitis and pulmonary fibrosis have an exudative diathesis as the basis for the development of lesions of the respiratory tract. Frequently this inability to retain water in the tissues is due to an inefficient supply of dextrose as evidence of hypoglycemia or ketosis.

As one studies associated disease of the sinuses and lower respiratory tract, he is impressed that the reaction of the mucous membrane to infection is much the same, whether in the nose or in the thorax. The tendency in the past has been to consider infections of the various parts of the respiratory tract as separate entities, each coming under the limited jurisdiction of a specialist for that locality. In the light of our present knowledge of the important relationship between sinus and chest infection, we should consider the respiratory tract as a whole. There is a definite overlapping of the responsibilities of the otolaryngologist, internist and Roentgenologist in dealing with this condition.

Let us now take up briefly some of the pitfalls of accepting too readily the probability of a causal relationship between the two infections. In our knowledge of the frequency with which the co-existing infection occurs, there may arise a tendency to jump to conclusions and make a diagnosis upon inferential grounds, without complete study. Experience of those working in this field establishes the fact that one examination by one specialist, whether he be Roentgenologist, internist or otolaryngologist, is not sufficient to substantiate the presence or absence of related sinus and bronchial infection. It may be unfortunate for the patient if we jump to conclusions as to

the presence or absence of sinus infection, based upon evidence seen in one Roentgen study. It may be equally unfortunate for him if we are too positive as to the nature of the chest infection after a single examination by clinical or X-ray means.

Wassan[9] cites a case of a child which he followed 10 years. During this time two examinations showed both maxillary sinuses with markedly thickened borders, while every other examination showed them excellently aerated. Another case which he followed seven years had definite recurrent sinus disease clinically; yet on several X-ray examinations the sinuses showed good aeration, with clear-cut borders.

Sinuses have been operated upon in a number of cases where they should not have been touched. Other causes may be responsible for peribronchial shadows and enlarged glands. Great care is sometimes required not to confuse glands in the chest, which are thought to be infected from the sinuses, with those in which the infection is due to tuberculosis.

In other instances, serious errors have been made by failing to grasp the fact that children with peribronchial tuberculous glands might coincidentally have infected sinuses; moreover, if we are going to accept the lymphohematogenous route from the sinuses as a possible pathway of infection, then other regions may act as a focus in the same manner as the sinuses. The tonsils and adenoids must not be overlooked. I recently had experience with a case in which the pediatrician, well aware of the frequency of associated sinus and bronchial infection, persisted in treating the nose by mass suction, although there was no demonstrable sinus infection. He ignored the presence of markedly enlarged, diseased tonsils and adenoids. Repeated examinations are frequently necessary by rhinologist, Roentgenologist and internist to arrive at a proper diagnosis. The one should be checked by the other before arriving at a positive conclusion. All three must work together to avoid serious error.

I know of no field in medicine which offers such great opportunity for a fine piece of constructive work by the method of co-operative group practice than does this field of respiratory infection.

## BIBLIOGRAPHY.

1. THOMSON, ST. CLAIR: Some of the Symptoms and Complications of Sinusitis. *Prac.*, 92:745, June, 1914; also, 4th Ed., Diseases of the Nose and Throat, p. 217.

2. SERGENT, EMILE: Histoire suggestive de quelques faux tuberculeux, diagnostics differentiels de la tuberculose pulmonaire et des affections des voies respiratoires superieures. *Bull. et mem. Soc. med. d. hop. de Paris*, 40:1424, July 28, 1916.

3. RIST, E.: Les principes de diagnostic rationnel de la tuberculose pulmonaire. *Press med.*, 24:305, 1916.

4. MULLIN, W. V., and RYDER, C.: Experimental Lesions of the Lungs Produced by the Inhalation Fluid of the Nose and Throat. *Amer. Rev. Tubercul.*, 4:683-687, 1920-21.

5. LARSELL, O., and FENTON, R. A.: Lymphatic Pathways from the Nose. *Arch. Otolaryngol.*, 24:696-713, 1936.

6. KERN, R. A., and SCHENCK, H. P.: Chronic Paranasal Sinus Infection Relation to Disease of the Lower Respiratory Tract. *Arch. Otolaryngol.*, 18:425-429, Oct., 1933.

7. CHURCHILL, EDWARD D.: The Pathology and Surgery of Bronchiectasis. (Thomas Dent Mutter Lecture L.), Dec. 1, 1937, at the College of Physicians of Philadelphia.

8. MULLIN, W. V.: The Accessory Sinuses as an Etiologic Factor in Bronchiectasis. *Ann. Otol., Rhinol. and Laryngol.*, 30:683, 1921.

9. WASSAN, W. W., and WALTZ, H. D.: The Relationship of Sinus Disease to Chest Disease in Children. *Radiol.*, 22:432-444, Jan.-June, 1934.

10. HODGE, G. E.: Relation of Bronchiectasis to Infection of Sinuses. *Arch. Otolaryngol.*, 22:537-547, Nov., 1935.

11. CLERF, L. H.: Bronchiectasis Associated with Disease of the Nasal Accessory Sinuses. *Arch. Otolaryngol.*, 6:28-35, July, 1927.

12. KISTNER, F. B.: Infections of Accessory Nasal Sinuses as the Cause of Chronic Non-tuberculous Bronchitis. *Northwest Med.*, 26:203, 1927.

13. MCLAURIN, J. G.: Chest Complications of Sinus Disease. *Ann. Otol., Rhinol. and Laryngol.*, 41:780-793, Sept., 1932.

14. WALSH, T. W., and MYER, O. O.: Coexistence of Bronchiectasis and Sinusitis. *Arch. Int. Med.*, 61:890-897, June, 1938.

15. GOODALE, R. L.: Analysis of 75 Cases of Bronchiectasis from Viewpoint of Sinus Infection. *Ann. Otol., Rhinol. and Laryngol.*, 47:347-359, June, 1938.

16. CAMPBELL, E. H.: Incidence and Significance of Sinusitis in Pneumonia. *Arch. Otolaryngol.*, 20:696-703, Nov., 1934.

17. CLERF, L. H.: The Inter-relationship of Sinus Disease and Bronchiectasis with Special Reference to Prognosis. THE LARYNGOSCOPE, St. Louis, July, 1934.

18. MULLIN, W. V.: Review of Sinus Chest Infections. *Ann. Otol., Rhinol. and Laryngol.*, 41:794-804, Sept., 1932.

19. ANSPACH, W. E.: Atelectasis and Bronchiectasis in Children. *Amer. Jour. Dis. Child.*, 47:1011, May, 1934.

20. REIMANN, HOBART A.: The Pneumonias, pp. 338-339, Sept., 1938.

21. SIPPE, CLIVE: Hypoglycaemia and Ketosis: Relationship to Chronic Antral Disease and Bronchiectasis. *Med. Jour. Australia*, 1:675, May 26, 1934.

259 South 17th Street.

## FUNCTIONAL VOCAL DISABILITIES.

MR. WILLIAM A. C. ZERFFI, New York.

From time to time laryngologists find themselves confronted by cases of laryngeal disabilities which are definitely functional in character and where treatment is contra-indicated. Vocal nodules, irritation of the vocal process, contact ulcers, to mention only a few, can be placed in this category. In such cases it is customary to prescribe vocal rest, and provided that the patient maintains a sufficiently long period of silence, the conditions tend to improve and may even disappear completely. This treatment, however, is merely in the nature of a palliative, for it is obvious that as soon as the patient begins to make use of his voice, the undesirable conditions are likely to return, and experience shows that this is usually the case. Needless to say, the physician will urge the patient to be careful in the use of his voice, to avoid forcing it, and so on, but suggestions of this kind are far too indefinite to be of any lasting value. For instance, the amount of effort which can be classed as abnormal is usually as unknown to the laryngologist as it is to the patient. Where singers are concerned, even those most severely affected would indignantly deny that they forced their voices, and yet the laryngologist often can see unmistakable evidence of improper use of the voice. Sometimes the suggestion is made that the patient take lessons in voice production as a means of relieving the difficulty, but experience also shows that in many cases the so-called expert in voice production is not sufficiently familiar with the physiology of the vocal organ to be capable of correcting bad vocal habits, so that both physician and patient often find themselves completely baffled.

Many years of study of the subject have convinced me that only a very radical change in the handling of such situations can be expected to yield a satisfactory solution.

The comparatively recently adopted viewpoint that the larynx is not a vocal organ, its primary function being that of a valve to keep food and liquid from entering the lungs, calls for a complete readjustment of all ideas hitherto enter-

Editor's Note.—This ms. received in Laryngoscope Office and accepted for publication, Feb. 10, 1939.

tained regarding the function of this organ in speech and song. Had the larynx been constructed solely for vocal purposes in the same manner as the eye or ear for their purposes, the problem would have been much simpler. But the larynx, as well as the throat, serve two purposes: The introduction of food into the body, as well as air into the lungs, which latter, during its exhalation, can be translated by the action of the vocal cords into speech or song. Hitherto study of the physiology of the larynx has been undertaken from the standpoint that barring pathological or other abnormalities, its action will be normally correct, and that such lesions as we are here considering are due to overuse or deliberate misuse of the organ. In other words, it is assumed that if the attention of the patient be drawn to these facts, a slight adjustment of his vocal habits will suffice to bring about a cure.

This, however, is by no means the case. Many bad vocal habits originate practically at birth and continue throughout childhood into adult life, so that by the time they have become powerful enough to cause serious trouble, they are so well established that a deliberate re-education of the throat muscles must be achieved before relief can be expected.

Superficial observers have taken for granted that the cry of an infant, being a spontaneous expression of nature, must therefore be a correctly produced sound. That is to say, that only after the child has been taught to imitate our artificial speech sounds, its naturally correct production will be upset. Let us, however, regard this matter from a different angle. The laryngeal muscles of a small infant must be extremely weak. Yet, despite this obvious fact, the infant is able to produce sounds of far greater volume than might reasonably be expected. It seems incredible that this volume of sound could be produced by its undeveloped vocal muscles, and the phenomenon can be explained only when it is realized that other more powerful muscles are involved. I refer here to the muscles of deglutition, which, as will presently be seen, can be used as aids in the production of sounds. As is well known, the larynx is closed during the act of swallowing, not only by the internal laryngeal muscles, but that certain external muscles, notably those of the tongue, assist in bringing about a complete closure. And it is by inducing a partial

closure of the larynx with the help of these external muscles and then forcing the breath out that the infant is able to produce so great a volume of sound. Once such habits of forced production are established, they find ready use in shouting and screaming, which involve an abnormal forcing together of the cords and an expulsion of the breath under considerable pressure. The degree in which such vocal habits affect the voice in adult life varies so greatly with the individual that it is impossible to generalize with any degree of accuracy. Much depends upon the temperament of the individual as well as upon the inherent ruggedness of the larynx. The majority of individuals go through life without much more than an occasional attack of laryngitis, but there are nevertheless a number of persons whose speaking voices cause them considerable trouble, particularly if they are obliged to make excessive demands upon them. Where singers are concerned, we find them liable to many vocal disabilities.

Experience in handling many kinds of vocal ailments leads me to the conclusion that functional vocal troubles are directly caused by the failure to inhibit the action of the extrinsic or swallowing muscles during the production of the voice. In other words, they are due to a carrying over of the faulty habits of voice production begun at birth and allowed to persist throughout childhood into adult life.

I suggest that a careful and thorough analysis of the action of the internal or true laryngeal muscles will reveal the fact that these muscles are literally incapable of acting in such a manner as would enable the vocal cords to be forced together. When used correctly for the production of tone, the cords are brought to a phonating position, first by the transverse arytenoid muscle, and pitch is raised by the combined action of the lateral cricoarytenoids, the thyroarytenoids, and the cricothyroid muscles. All these muscles are subconscious in their action, responding to the desire to produce a certain pitch, but are removed from direct control. Needless to say the subject to be examined is directed to sing a tone, not requested to approximate the vocal cords. On the other hand, the act of swallowing can be induced or inhibited at will, movements of the tongue likewise, though these actions are so habitual as to seem to be subconscious. It is generally

accepted that the occurrence of vocal nodules is due to a continued irritation at the junction of the anterior with the middle third of the cords. If the action of the internal laryngeal muscles be carefully followed, it will be found that these muscles are incapable of acting so as to force the anterior portion of the cords together. The conclusion is therefore unavoidable that the pressure necessary to produce the nodules must come from a source other than that of the true laryngeal muscles.

In analyzing vocal troubles it seems frequently to be overlooked that the valvular action of the larynx is used for purposes requiring vastly more effort than that of voice production; for instance, to lock the breath in the chest when the arms are used for lifting weights or any similar exercise. In such cases where great effort is required, use is made of the external muscles to assist in bringing about a forcible closure of the larynx. In incorrect voice production unusually loud tones or tones of high pitch are produced also by the assistance of the external or swallowing muscles.

While it might seem at first thought that recognition of such faults of production would involve a deep and lengthy study of voice production such as would lie outside the activities of the laryngologist, actually the detection of the interference of the external muscles with the true laryngeal muscles is a relatively simple matter. In actual experiment, if the finger be placed above the larynx in the angle of the chin, swallowing will reveal the cooperation of the external muscles. If the finger be returned to the original position and the attempt to speak or sing is made, any muscular movement akin to that induced by swallowing reveals an unnecessary involvement of the external muscles. That is to say, the vocal cords are being brought together with greater force than their true muscles can develop and consequently there is danger of irritation of the free edges of the cords. In addition, the normal action of the vocal muscles is being disturbed, with the result that they become weakened through lack of use. When this has occurred, the patient is more than ever dependent upon the help furnished by the extrinsic muscles, being literally in a vicious circle. This method of finger palpation has been used by Dr. Elmer L. Kenyon, of Rush Medical College, who states: "The value of the observation

of the action of the movements of the thyroid cartilage by finger palpation cannot well be overstated."

It need hardly be said that the correction of such vocal difficulties as are caused by the interference of the external muscles is something which lies outside the province of the laryngologist, but an awareness of the facts would enable him to more speedily diagnose functional vocal disorders and also to judge whether the patient is receiving help from whatever source he may seek it in the field of voice production.

The greatest need in the vocal world today is an intimate cooperation between the laryngologist and the teacher of voice production, a co-operation which, except in rare cases, literally does not exist. As Dr. Robert F. Ridpath, of Philadelphia, speaking at the 1937 Session of the American Medical Association in Atlantic City, said: "There are few laryngologists who have more than a meager knowledge of the art of vocal training and there are fewer vocal teachers who have any knowledge of the anatomy, physiology or the phenomenon of voice production. It is mainly this unfamiliarity or ignorance of the subject at both sources that is responsible for a great many vocal tragedies."

31 West 84th Street.

---

## BOOK REVIEW

**Les Maladies du Larynx Clinique et Therapeutique.** By G. Canuyt, Professor de Clinique Oto-Rhino-Laryngologique a la Faculte de Medecine de Strasbourg avec la collaboration de M. M. Truffert, Ancien Chef de Clinique a la Faculte de Medecine de Paris, etc. Eight hundred fourteen pages with Index. Ten plates in color and 326 figures. Paris VIe: Masson et Cie, Editeurs, Libraries de L'Academie de Medecine, 120 Boulevard Saint-Germain. 1939. Price 270 fr.

This book easily represents the most elaborate and thorough modern work on the larynx and its diseases. The chapters on radiography and tomography of the larynx represents the latest thoughts on these subjects. Other chapters, equally thorough and interesting, discuss the various diseases of the larynx. Of particular interest are the chapters on the tumors and malignancies of the larynx. The discussion of the disorders of speech, the speaking and singing voice add to the value of the book. The very fact that only one or two American authors are quoted serves to give to the American public a chance to become familiar with European views on laryngeal conditions. It is sincerely to be hoped that an English translation will be forthcoming, and to those who are particularly interested in the larynx this book cannot be too highly recommended.

F. E. LE J. AND P. J. B.

# NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

*Meeting of May 3, 1939.*

**(a) — Primary Carcinoma of Middle Ear and Mastoid.**

**(b) — The Endaural Approach to the Mastoid.** Dr. Clarence H. Smith.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. G. A. ROBINSON: In the management of malignant tumors of the middle ear, we feel that preoperative radiation, surgery and postoperative radiation should be employed. The same general plan of treatment is also used in the treatment of carcinoma of the nasal sinuses.

The prognosis depends upon the type of growth and the extent of involvement.

Preoperative radiation kills tumor cells, lessens the secondary infection and causes fibrosis of blood vessels, thereby decreasing the amount of hemorrhage at the time of operation. Surgical procedure removes the necrotic tumor tissue and allows for proper drainage. Postoperative radiation will have its effect on any viable tumor cells that remain after the operation.

I should like to emphasize the importance of giving the radiation in small, divided doses according to the Coutard technique in order to obviate as far as possible osteomyelitis and radiation necrosis.

DR. THOMAS J. HARRIS: First, I want to congratulate Dr. Smith on the splendid presentation he has made. I think it is one of the most interesting and valuable I have heard in a long time. I want to speak particularly of this case of primary carcinoma because I had under my care a number of years ago a case that I diagnosed as squamous cell epithelioma of the external auditory canal. It is questionable whether the diagnosis was correct.

The patient was a clergyman, age 30 years, who came complaining of pain in the ear with a discharge. At the early examination there was no sign of a growth, but very shortly afterwards it was possible to detect endaurally a mass of very easily bleeding tissue. The patient was taken into the hospital and we endeavored to remove the tumor by the postauricular route, but immediately upon attacking the growth there was such enormous bleeding that it was impossible to do anything but remove a specimen and then pack the wound and close it. The report from the St. Luke's Hospital laboratory was that we were dealing with a primary squamous cell epithelioma of the external canal, and I so reported the case before the New York Otological Society. But a short time ago Dr. Risch, a member of this Section, was studying the literature and came across the case I had reported, and he felt there was some question whether we were not dealing with a sarcoma. After the failure to remove the growth by radical procedure, we resorted to radium. The patient was put under treatment at the Memorial Hospital and had a sufficient amount of treatment to cause the growth entirely to disappear. It remained away for more than two years, when it returned with involvement of the intracranial structures. There was paralysis of the facial muscles and of the recurrent laryngeal nerve. He survived for some time, and in spite of the laryngeal involvement was able to preach for several years, and then he succumbed, due to the advancement of the growth. I feel that this case could be added to the series Dr. Smith has reported. The pain, discharge, frequent hemorrhages

from the ear and involvement of the nerves all point to the fact that we were dealing with a primary epithelioma.

DR. CLARENCE H. SMITH: The amount of bleeding in my case was not abnormal. The radiation treatment had cut down the size of the tumor very appreciably. The bony changes were marked, however.

In think the radiation therapy had a great deal to do with this man's recovery.

**Acute Mastoiditis Masked by Sulfanilamide.** Dr. Samuel Rosen.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. FREDERICK M. LAW: This description of a typical case of mastoiditis masked by the administration of sulfanilamide is particularly interesting to me. I am speaking now entirely from a radiographic point of view. Since this drug has been used, we have noticed the series of symptoms which Dr. Rosen has described; and I am warning you now, as a Roentgenologist — in any case which you refer to a Roentgenologist for X-ray diagnosis, specify on your request whether or not sulfanilamide has been given. The drug makes a great difference in the appearance of the films. It has some peculiar effect on the contents of the cells — in other words, makes them translucent to the X-ray. A film of acute mastoiditis shows opacity, but after the administration of sulfanilamide it becomes slightly translucent and more nearly resembles a normal mastoid. Sulfanilamide does not influence the progress of the disease if it has attacked the bone before you give the drug. Your film in that case will show translucency, but properly made films will indicate bone changes; and when you have given the drug and your films show a bone change, there is no use waiting, you might as well operate. The disease progresses just the same as though you had never given sulfanilamide.

This observation is recent, but it has proven true in many, many cases, so do not trust a Roentgenologist's report on a mastoid film unless you have given him notice of the use of sulfanilamide.

DR. JACOB L. MAYBAUM: Dr. Rosen has reported a picture resulting from the administration of sulfanilamide in an otitic infection which is getting to be more and more recognized as being almost commonplace. It is true that there are members, particularly of the pediatric specialty, who scoff at our constant reference to the fact that sulfanilamide should be used with considerable discrimination in otitic infection. Two years ago, or a little more than that, I saw for the first time in the January, 1937, issue of the Archives of Otolaryngology a case reported by Smith and Coon of a masked ear picture in which sulfanilamide had been given. Following a mastoid operation, a septic temperature was noted, but blood cultures were repeatedly negative. The child was euphoric. Then it occurred to someone that it might be a good plan to do a Tobey-Ayer test to establish, if possible, the presence of a thrombosis. To the great surprise of the onlookers, the spinal fluid was very cloudy, revealed a high cell count and cultured out positive for streptococcus hemolyticus. These cases are reported again and again, so that we have formulated for ourselves — with a proviso that we have the privilege of changing our viewpoint — when and when not to use sulfanilamide. We feel at present that it is inadvisable to use sulfanilamide in a case that looks in the least suspicious of developing a mastoiditis. We will not give it in an acute suppurative middle ear infection. It may possibly be of value in an early otitis media before suppuration has taken place. We do not give sulfanilamide following mastoidectomy or before, if a patient is running a temperature and we suspect a possibility of sinus thrombosis. When do we give sulfanilamide? We reserve it only for otitic complications in those instances in which a diagnosis has been made, particularly in the case of meningitis, or following a sinus thrombosis operation, and in cases of sympathetic meningitis before

and after removal of the focus. We advise that if used in acute otitis, we should observe carefully the clinical course and otoscopic picture; secondly, that the drug should be discontinued for a time to determine if a flare-up of signs and symptoms occurs, as in the case report of Dr. Rosen.

What do we mean by masking the symptoms by sulfanilamide? The picture resembles that of a pneumococcus type III otitic infection. The middle ear, which had been discharging, will stop discharging, the drum is thickened and the landmarks are obscured, tenderness disappears, and then, if the drug is stopped, there may result definite signs of a mastoid involvement or an intracranial complication. The drug unquestionably also changes the picture of an otitic complication. We see clinical varieties of meningitis such as we never observed in the past — cases practically symptomless, with temperature down, and yet on doing a lumbar tap all the diagnostic evidence of a bacterial meningitis can be demonstrated on examining the spinal fluid. Without being too dogmatic, I should say that one should be very cautious in the use of sulfanilamide in otitic infections. It is possible that we may change our viewpoint as our experience and that of others in the use of this remarkable drug increases.

The observation made by Dr. Law is very interesting to me because I have observed in a number of cases in which sulfanilamide had been given, similar findings — that is, the X-ray signs are practically nil, and yet by this time we had made a clinical diagnosis of the presence of a mastoiditis.

Dr. Francis W. White: I have been fortunate in being able to follow a number of films with Dr. Law in a case which Dr. Harris will probably report later. When we were looking at them, Dr. Law would say: "This is a 2+ unless sulfanilamide has been given, otherwise it is 4+." The patient may be up and around the ward, wanting to go home, be operated on and a short time later require a sinus operation.

*(To be continued in a succeeding issue.)*